INFANTILE PARALYSIS

IN

MASSACHUSETTS,



1907-1912.

TOGETHER WITH REPORTS OF SPECIAL INVESTIGATIONS IN 1913, BEARING UPON THE ETIOLOGY OF THE DISEASE AND THE METHOD OF ITS TRANSMISSION.



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THE STATE BOARD OF P[illegible]

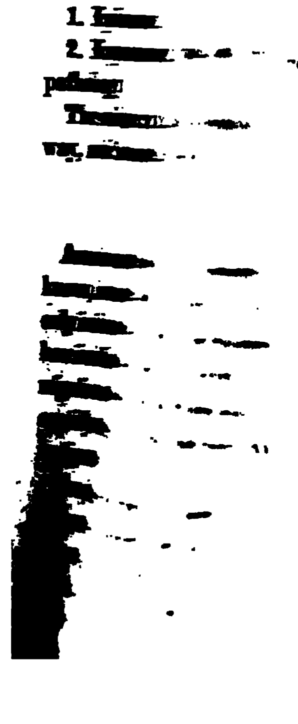


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man Naturalists and Physicians in 1838. Before this, however, Dr. John Badham of Workrop, Eng., had published four well-authenticated cases of infantile paralysis in children of two years of age which occurred at about the same time and in the same locality. Heine, in his monograph of 1860, had already noted the possibility of the contagious or epidemic character of the disease.

The next to describe the disease were Rilliet and Barthez, French pediatricians, but Charcot's description of 1870, with full autopsy reports, is the most important. In the summer of 1887, O. Medin noted the first epidemic of the disease in Stockholm.

Harbitz and Scheel state that in Norway there were 18 cases of infantile paralysis in 1903, with 6 deaths; in 1904 an epidemic of 41 cases and 6 deaths, and a second one of 20 cases and 6 deaths.

In 1905 the disease became more serious and invaded a great many districts. Altogether, 719 cases with 111 deaths were reported in 1905, and 334 cases with 34 deaths in 1906, giving a mortality of 13.8 per cent. in two years. The mortality varied according to the frequency of the occurrence of abortive cases.

Early in the summer of 1907 there began an epidemic of poliomyelitis (1) in and about New York City, so that in one hospital there were treated 138 cases. The occurrence of the epidemic suggested a mode of infection through the intestinal tract, in that many of the cases were preceded by diarrhœa and dysentery. Out of 100 cases diarrhœa and vomiting occurred in 19 per cent., vomiting and constipation in 11 per cent., diarrhœa alone in 11 per cent., constipation alone in 11 per cent., and vomiting alone in 11 per cent. In support of the theory of the epidemic nature of the disease a comparison of 1906 with 1907 showed that in 1906 there were but 36 cases, while for the corresponding time in 1907 there were 387 cases, of which 216 were males and 171 females. In this epidemic there were many cases beyond the infantile period of life.

Starr (2) reports the same epidemic more fully, stating that there were probably over 2,000 cases, extending along Long Island Sound into Connecticut and up the Hudson. The disease began to show itself about May, 1907; the number of cases increased rapidly during June and July, and the epidemic reached its height in July and August, and a few were then reported until December, 1907. The mortality was about 6 to 7 per cent. The summer was not an unusually hot one, but the rainfall was about one-half the usual amount. Other infectious diseases were not particularly prevalent.

The onset of the disease was acute, the paralysis developed on the

third or fourth day, appearing with considerable suddenness and at its maximum extent from the beginning. Pain was a marked symptom in almost all of the cases, and was referred to the muscles involved or to the back. The acute onset usually subsided in the course of a week or ten days, and a state of improvement was noticed beginning at the end of the second to the fourth week. In many cases complete recovery occurred. In these cases the paralysis was never very intense; in others it was merely a sense of fatigue and unwillingness to use the muscles. These were classed as "abortive" cases. The high mortality of 7 per cent. was unusual, as death is a very rare occurrence in sporadic cases.

Starr states that epidemics of infantile paralysis are more common than has been supposed, and has collected a list of 44 such epidemics, which he presents as follows in order of their occurrence: —

Leegaard (3½), in Finland, was thought to have been the first to describe an epidemic of poliomyelitis, at least in Europe, his report covering 13 to 18 cases. It was then found that the disease had been clearly described in 1868 by A. C. Bull, a county physician in Norway, under the title of "meningitis spinalis acuta." Bull reported 14 cases and 5 deaths; 12 cases occurred in children between four and ten years of age, 1 in a girl of fifteen and 1 in a man of twenty. He also mentioned the so-called abortive cases.

Colmer (3) reports 11 cases in West Feliciana, La., in the fall of 1841, where all the cases were under two years of age, and where all recovered.

Bergenholtz (4) reported an epidemic of 13 or 18 cases in 1881, in the town of Umea, North Sweden.

Cordier (5) observed 13 cases in June and July, 1885, in the town of Saint Foy, near Lyons, France. Other cases were seen and heard of in the vicinity; 4 died of respiratory paralysis.

Oxholm (6) reported an epidemic in Mandel, Norway, in July, August and September, 1886, of 9 cases without mortality.

Medin (7) observed an epidemic between May and November, 1887, in Stockholm, of 43 cases, with 3 deaths. Medin was the first to call particular attention to the epidemic occurrences of infantile paralysis at the tenth International Medical Congress in 1890.

Putnam (8), in the summer of 1893, observed an unusual number of cases in Boston, Mass., where 26 cases were observed in the months of August and September, when the usual number seen was about 6 to 8.

André (9) saw 4 out of 9 cases observed at Saint Girons, near Toulouse, France, and its two adjacent towns, Cescan and Seix, in July and August, 1893. They all occurred in infants under 3, and all recovered.

Caverly (10) reported the first serious epidemic in this country, which

occurred in Rutland, Vt., between July and September, 1894. Rutland is in a valley, and it is interesting to note that in a large number of these epidemics the site of the occurrences has been near a stream in a narrow valley, during the summer which has been extremely hot and rather dry. This was the case in Rutland. He records 126 cases with 18 deaths. It was noticed in this epidemic that horses, dogs and chickens were also affected.

Pieraccini (11) noted a small epidemic in Monte Spertoli, near Florence, Italy, a village lying on the side of a mountain, in a high altitude and a dry locality. This epidemic occurred between June 23 and Aug. 10, 1895. There were 7 cases, all of which recovered.

Bucelli (12) reported a number of cases in the Ravicca quarter of Genoa in 1895. He also adds that an epidemic prevailed at the same time in Arenzano, a suburb of Genoa.

Medin (13) observed an epidemic in Stockholm in the summer of 1895. There were 20 cases seen between June and September; all had fever and the majority had the legs affected. This epidemic occurred in the latter part of the summer and early fall, at a time when infantile diarrhœa was quite prevalent in Stockholm.

Altman (14) observed an epidemic of 18 cases at Port Lincoln, South Australia, during the months of March and April, 1896, the hot and dry months in Australia. This appears to have been the first epidemic on record in Australia. Port Lincoln is a small town of not over 1,500 inhabitants. The cases were all over three years of age, and none died.

Pasteur (15) observed at Much Hadham, in England, in July, 1896, 7 cases in one family living in the same house, all attacked within ten days. The neighborhood was a healthy one, and there were no particular circumstances indicating in any way the cause of the disease. The children ranged from one and one-half to eleven years of age. There were no other cases in the neighborhood.

Taylor (16) saw in July and August, 1896, 7 cases at Cherryfield, Me. This disease appeared to be infectious, as the second patient was a brother of the first, the third, a cousin of the first and second, and the fourth was visiting in the house where the first 3 patients were affected. There were also 2 cases occurring at the same time in a neighboring town.

Bondurant (17) reports 15 cases occurring in Greene County, Ala., during the summer of 1896, both whites and negroes being affected. There were no deaths. Malaria was present at the time.

Buzzard (18) calls attention to an unusual number of cases of infantile paralysis in a small district of London, in September, 1897, and gives

details in 11 cases. All were children under five, except one boy of fourteen. In two instances the patients were sisters, or a brother and sister. There were no deaths. Several others were heard of at the time but not seen.

Taylor (19) saw a rather large number of cases at the Hospital for Ruptured and Crippled, in New York, in July, 1897.

Pleuss (20) records 4 cases which had been seen in Kiel, between June and September, 1897. These might be accounted for as sporadic cases.

Newmark (21) of San Francisco records a small epidemic occurring in the town of Le Grand, Merced County, Cal., in June, 1898. It is a village of 49 people; none of the patients died. Four children were affected, the first and second being brothers, the third a playmate of the first and second, and the son of the doctor who attended them. They were all boys between eight and ten years of age.

Packard (22) records the fact that during the summer at Royersford, Pa., there were two cases developing in the same house, within three days of each other, in a brother and sister. He considers this observation as of importance in supporting the infectious nature of the disease.

Auerbach (23) reports a small epidemic occurring in Frankfort-on-the-Main between May and December, 1898. There were 9 cases observed and several others heard of. In one case there was a recurrence of the disease in August after the first attack in June. This case is noteworthy for the fact that one attack usually confers immunity.

Zappert (24) reports an epidemic which occurred in Vienna in 1898. Two hundred and eight cases were observed, and these occurred chiefly in the summer, between June and November. No cases were observed where other children of the same family were attacked.

Wickman (25) observed 54 cases during an extensive epidemic in Stockholm in the summer of 1899; 3 cases died. The majority of the cases occurred in July, August and September, and almost all the patients were under five years of age. In 3 different houses 2 cases of the disease occurred, and in several families 2 or 3 cases were observed among cousins, brothers and sisters. Wickman noticed that the disease was not generally spread throughout the city, but was localized in certain parts; so that the cases tended to be grouped. In one instance there occurred a case in a house from which a family moved; a second case developed in the family which moved into this house soon after it was vacated by the first family. This he considered good evidence as to the infectiousness of the disease.

Leegaard (26) in Bratsberg, Norway, observed 54 cases between April

and November, 1899. All ages were affected, but the majority were children under eleven; 2 cases died.

Chapin (27) gives the details of 7 cases seen by him during an epidemic in Poughkeepsie, N. Y., in July and August, 1899; 30 or 40 cases were seen by physicians during that summer. The majority of the cases made a complete recovery, a fact which has been noticed in a number of epidemics, and has a distinct bearing on the prognosis.

McKenzie (28) describes 17 cases occurring in Washington and Amenia during the summer and autumn of 1899. These are small towns near Poughkeepsie, N. Y., and the epidemic coincided with the one described by Chapin. Three of the 17 cases died within four days of the onset.

Painter (29) saw 52 cases between June and September, 1900, in Gloucester, Mass., 38 of these were under his care. They were in children between thirteen months and ten years of age; there were no deaths. In some instances two members of the same family were affected.

A. M. Woods (30) reported an epidemic of 55 cases in San Francisco, in March and June, 1901. The weather was damp and cool. Most of the cases occurred in infants. There were no deaths.

Wickman (31) observed 20 cases in Göteborg, Sweden, between July and November, 1903; 3 were sisters; 1 case died.

A. Lorenzelli (32) reported 26 cases in Parma, Italy, between March and September, 1903. The majority of cases were under five.

Blackhall (33) reported 6 cases in Queanbergan, New South Wales, during an extensive epidemic in December, 1903, and January and February, 1904, which is the hot season in Australia. Two cases were sisters, 2 were cousins; none of the cases was fatal.

Litchfield observed 25 cases at Sydney. The summer was more wet and cool than usual. During the summer there was an extensive epidemic of gastrointestinal influenza. There were no deaths.

R. B. Wade observed 34 cases at Stanmore, near Sydney, all cases being under seven years of age. All cases had a febrile onset with vomiting, diarrhœa and severe sweating; there were no deaths. Some permanent paralysis was left in the majority of cases. In several instances two or three members of the same family were affected. Some would have a permanent paralysis, while others in the same family would suffer from malaise and general weakness, but make a full recovery. This was the second Australian epidemic.

B. B. Ham (34) reports an epidemic in Brisbane, Queensland, from October to December, 1904. One hundred and eight cases were reported with almost all patients being under the age of ten; 4 cases died. This was the third Australian epidemic.

Nannestad (35) observed 41 cases in Hvalen, Norway, between June and October, 1904. Most of the cases occurred in infants. No case died.

Platon (36) observed in the same year, in another part of Norway, a small epidemic at Aafjorden in August. There were 20 cases and 6 deaths.

Geirsvold (37), in Norway, reported the most extensive epidemic hitherto reported, which occurred in Norway and Sweden in the summer of 1905. He observed 437 cases in the neighborhood of Trondhjem between April and October. There were 67 deaths. It was apparently possible in this epidemic to trace the extent of the disease by contact from one village to another, and from house to house. There was a difference of opinion among the physicians as to the contagiousness of the disease, and some laid it to the condition of the water supply, and pointed out that the disease was more prevalent in damp places surrounded by swamps.

Wickman (38) described in detail 1,031 cases occurring between May and October, 1905, in the southern part of Sweden. These cases were distributed through a large number of villages adjacent to each other, and Wickman believes that his careful investigation proves the contagious character of the infection. Many cases occurred in the same house, and in many instances two or more members of the same family were affected. In one town 16 or 18 cases originated from a single schoolhouse. In the majority of cases contact between cases could be traced. New tenants of houses vacated by patients suffering from the disease were affected also. Wickman investigated the epidemic with great care, especially the question of contagion and mode of extension, and from his evidence it is hard to escape the conclusion that the disease is actually contagious, and that like scarlet fever it can be carried through the medium of a third person. Dogs and other animals were likewise affected in this epidemic.

Wickman (39) saw a smaller epidemic in Sweden in 1906, reporting 50 cases.

Lundgren (40) noted an epidemic at Vardo, Norway, in the summer of 1905. He records 403 cases, occurring chiefly in children between six and fifteen years of age. The disease was apparently contagious, as many cases were traced where the contagion seemed evident. Ten per cent. of the cases died, — a very high mortality. Permanent paralysis of some type was left in 25 per cent. of the cases.

Terriberry (41) reported an epidemic of 50 cases which occurred at Ridgway, Pa., in the summer of 1907. The geographical conditions of

this epidemic were of interest. Ridgway is situated in a valley in the Allegheny Mountains, about 16 miles in length. The first case appeared at Elk Creek, where 12 cases were seen. Two weeks later cases appeared at Ridgway, 9 miles below Elk Creek, and in that town 30 cases eventually appeared. One week after the cases appeared in Ridgway, cases developed in Scotland Mills, 9 miles below Ridgway, and there 8 cases developed. It was evident that the infection extended down the valley in a definite direction, and that the incubation was from one to two weeks. Four cases out of the 50 died. They were all children, and in every instance 2 or 3 cases appeared in the same family.

Griffin (42) reports 20 cases occurring in Oceana County, Mich., in the summer of 1907, chiefly in infants. In proportion to the population there were ten times as many cases in Oceana County as in the New York epidemic.

S. M. Free (43) reported an epidemic which occurred at Dubois, Pa., in the summer of 1907. Over 100 cases were observed in the Allegheny Mountains about Dubois. There were only a few deaths. Pigs and chickens were also affected.

Ordinarily the mortality of poliomyelitis in sporadic cases is very low, but in epidemics it varies from 6 to 10 per cent. Fatal cases occur between the fourth and tenth days, the paralysis extending to the respiratory muscles. It was also demonstrated that about 25 per cent. of the cases made a complete recovery, whereas this is not true in sporadic cases.

Hamilton (44) reported three epidemics of poliomyelitis, all more or less extensive, occurring in Minnesota in 1908; at Hibbing 16 cases were reported; at Northfield, 30 cases, and at Barnum and Moore Lake, 45 cases. There were also numerous isolated cases throughout the State. All epidemics came in the autumn and disappeared after the onset of cold weather. Thirteen deaths were reported out of 150 cases. There was no evidence found that the disease was infectious or contagious. It was also reported that an identical disease occurred among colts and young horses at the same time.

Collins and Romeiser (45) reported the New York epidemic of 1907, which began in July and ended in October, there being as many as 1,200 to 1,500 cases. In this epidemic 1 case in 10 or 11 made a good recovery, and about 1 in 20 recovered absolutely. In the analysis of 500 cases there were 202 males and 114 females affected. The dangerous age was between one and three.

Lovett (46), in the report of the Massachusetts State Board of Health on infantile paralysis for 1909, speaks of the various epidemics, especially

one of 140 cases in Cuba, in 1909, and states that this was the first epidemic reported from the tropics. Apparently the disease did not exist in Cuba before 1907. It seemed possible that the disease was imported to Havana from New York. He states that in the five years previous to 1909 about 8,054 cases occurred, of which the United States contributed 5,514, the bulk of these being in the northern States. He states that in 1909 Massachusetts suffered as much from the disease as any country in Europe. As many cases were reported in Massachusetts as in the two other most severely affected States in the Union (Nebraska and Minnesota) taken together. It occurred generally in river valleys, and a case rarely occurred in a town without one or more cases in contiguous towns. It was relatively more prevalent in small towns than in the larger towns and cities. The total number reported was 923. There was no deficiency in the rainfall, nor was there any connection between the incidence of the disease and the high temperature. There was found to be no corresponding infection of domestic animals as reported in some other epidemics. No child, of 150 cases carefully investigated, living on breast-milk alone was affected. Three hundred and sixty-three males and 263 females were affected, with the period of greatest infection seen between the ages of two and three years. Seven per cent. of the cases occurred in the first year of life, 71 per cent. occurred in the first five years and 87 per cent. of the cases in the first ten years of life.

Krause (47) reported the epidemic which occurred in Westphalia in 1909. The entire number of deaths among 436 cases of the Province of Arnsberg numbered 66; the youngest child attacked was four and one-half weeks and the oldest fifteen years. Most cases occurred in the second year of life. Girls were affected more than boys. In over 90 per cent. of the cases symptoms referable to the stomach and intestines were noted at the beginning of the disease, and in two-thirds of the cases the children were attacked with severe diarrhœa, rarely with vomiting, while in one-third constipation was present. In many cases other members of the family had diarrhœa. The paralysis affected the limbs in the following order: one leg or single group of muscles; one arm; both legs; one-half of face; both legs and one arm; and lastly paralysis of the bladder which occurred only with coincident paralysis of both legs. In the fatal cases respiratory paralysis seemed to occur regularly. Krause distinctly advocates the infectious character of infantile paralysis. The occurrence of groups of cases was plainly established. A remarkable mortality among chickens coincident with the disease was established.

Shidler (48) reports a number of cases occurring in the epidemic in

Nebraska in 1909. He shows by some striking facts how apparently the disease spread by infection from town to town and from family to family. He analyzes 60 cases carefully. The evidence on the infectious nature of the disease is most striking. He showed how a young man working on a threshing crew with 7 other men contracted the disease. These men all drank out of the same jug, worked together, ate together, and all developed the disease. The disease spread from Benedict to Marquette and elsewhere in the following manner: A child came into Benedict from near Polk, Neb., and was taken ill; it returned to the country, where the disease was diagnosed as epidemic poliomyelitis. The twelve-year-old daughter of the family next had the disease. Several weeks later the son-in-law of the parents of the affected children came from Marquette, Neb., with his infant son and visited over night. In five days the infant came down with the disease, this being the first case in Marquette. A child of another family came in from the country to the house of the above-mentioned family in Benedict about a month later, not having been allowed by its parents to see any other child all summer. She stayed one night in this house, returning home in the morning. In five days she became ill, and after three days developed paralysis of both legs. Other striking instances of apparent contagion are given.

McClanahan (49) reports more fully the Nebraska epidemic of the summer of 1909. There were slightly over 1,000 cases, with about 137 deaths. The first case was reported in March, and only 20 cases after November. Four-fifths of the cases occurred during July and August. These months were unusually warm and dry. Fifty-five per cent. of all cases occurred in Polk and York counties, with a total population of 31,000 people. It was a disease of the rural districts. A 4th of July celebration was held in Stromsberg, Polk County, and within two weeks after that the disease spread like a wave over the adjoining country. Quarantine was established with excellent results. The fatal cases were nearly all of the bulbar type of paralysis. Constipation was the rule; diarrhœa, the rare exception; vomiting was rare. The disease seemed to have an inhibitory effect on the intestines. The extreme age limits were four months and sixty-seven years. The oldest fatal case was thirty-seven.

Jacolyn Manning (50) reports an epidemic of 352 cases which started in 1908 in Eau Claire County; 167 of the total cases occurred in that county. She gives a mortality of 15.3 per cent. She noted a rash in every case she saw in the febrile stage. Twenty-five cases became ill during the time of, or immediately following, cases in the same house. The maximum number of cases occurred in August and September, at a time when the rivers were lower than they had been for years.

Lovett (51) in a second paper reports the results of the Massachusetts State investigation for 1908. One hundred and thirty-six cases were reported in 1908, as against 234 in 1907. Localities severely affected in 1907 were comparatively immune in 1908. Most of the cases occurred in the months from July to November. The majority of the cases occurred in children between the ages of one and two years.

Emerson (52) states that nearly one-half of all cases reported in Massachusetts in 1909 occurred in the western half of the State, in adjacent river valleys. The disease is an infectious one. At the most it is mildly contagious. The marked digestive disturbances suggest the stomach as the portal of entry.

Kramer (53) reviews recent epidemics and states that the mortality is higher than had been supposed, and also the number of cases of complete recovery. In the Breslau clinic 196 cases were seen ranging from infancy to thirty years. The summer months showed the largest number of cases.

Koplik (54) reports the New York City and State epidemic of 1909; there were about 1,200 cases. There seemed to be no etiological factors in weather, water or milk supplies, city cleanliness or localization of cases. He speaks of the atypical character of many of the cases, and divides them into three groups, namely, cerebral, neuritic and classical, the latter being the usual common type. Intestinal disturbances were present in the great majority of cases. He concluded that we were dealing with an infection that may attack the white matter and axis cylinders as well as the gray matter of the cerebrum, medulla and cord, and may produce typical acute infectious neuritis as well as anterior poliomyelitis.

Kerr (55) reports 53 cases he observed, — a part of the New York epidemic of 1909. Fever, diarrhœa and vomiting were the prominent symptoms at onset.

Wollenweber (56) reports 31 cases personally observed in Dortmund in 1909. His views corroborate those of Krause in that it was an infectious disease with diarrhœa, apparently contagious, with an incubation period of from two to fourteen days.

Langermann (57) reports 4 sporadic cases in the village of Garbenteich, from September to November, 1909. It became epidemic by transportation from Marburg, where it existed in epidemic form. Eight cases were observed, 2 of them abortive and 3 fatal.

Reckzeh (58) states that so far, in 1909, 500 cases have occurred in the district of Amsberg. The mortality in Bochum was 18.2 per cent. There were no essential differences in this epidemic from the others reported.

Ferreira (82) states that poliomyelitis made its appearance in Brazil towards the close of 1909. Thirteen cases in infants were observed during the summer at Sao Paulo and a number in Rio Janeiro.

Treves (59) reports an epidemic of 8 cases in Upminster, Eng., a town of 1,700 people. There had been no isolated cases in the town for several years. The weather was hot and dry, but not excessively so. The legs were usually affected.

Eichelberg (60) reports 31 cases observed in an epidemic in Hanover, in October and November, 1909, — one branch of the first great German epidemic. The onset in 16 cases was with intestinal symptoms, and in 14 they were referred to the air passages. Paralysis was very severe at first. There were 7 deaths, all from respiratory paralysis. The ages of the cases varied from six months to eight years, 16 occurring in the second year. There was usually no evidence of connection between the cases.

Peiper (61) reports the cases occurring in Vorpommern in 1909. There were 51 cases recognized, and the epidemic extended from July 15, 1909, through the autumn, especially in October, when 31 cases occurred.

Poliomyelitis prevailed in epidemic form in Kansas (79) during the summer of 1909. The disease had appeared before in sporadic form in the State, but this was the first epidemic, there being somewhat over 100 cases. The first cases were reported in the latter part of July, and the height of the epidemic was reached in August and September, with a few cases as late as the last of November. The season was unusually hot and dry, particularly in the two counties where the largest number of cases occurred. With the appearance of cold, wet weather the disease ceased. In only 9 families in the Kansas epidemic, out of 58 whose histories were noted in regard to this point, was there more than 1 case in a family, although every family but 8 had more than one child in the family. In 61 case reports there were 18 deaths, giving a mortality rate of 29.5 per cent., which is very high. No abortive cases were reported, although they probably existed and were not recognized. The paralysis attacked both legs, or both arms and legs, with greater frequency than other parts, and usually made its appearance on the second or third day. There were no other points of interest noted which would enable one to draw any definite conclusions as to the method of infection. The majority of the cases were between one and three years, the oldest being forty-three years and the youngest six months. No method of contagion could be found, and the author does not consider the disease contagious. The mortality was 11.7 per cent. One-third of the cases had one extremity affected, facial paralysis in 1 case, and 10 cases were reported as relatively recovered.

Netter (63) reports the epidemic of 1909 in Paris, and concludes that the disease is contagious and that the cases should be isolated as other contagious diseases.

This is a very interesting document (64) published by the State of Washington and detailing the cases which occurred in that State in 1910. The plan of report was copied from that used by the Massachusetts State Board of Health. There is an interesting preliminary historical study. There were 397 cases reported. Their investigations showed that there was practically an even distribution of cases between the larger cities and the smaller cities and the rural districts. The incidence of the disease was greatest between the ages of two and three years. The mortality of the reported and investigated cases, namely, 331, was 8.4 per cent. Fever and constipation were the usual early symptoms, also pain, tenderness and vomiting were reported with about equal frequency. Eighteen cases had retention of urine, which condition had also occurred in other epidemics. There was no coincident paralysis among the domestic animals. There were many striking instances of possible contagiousness reported. There seemed to be 23 cases of apparent transmission from person to person, by direct or indirect contact. The seasonal prevalence showed that the majority of the cases occurred in the summer months, as was true of all other reported epidemics.

House (78) saw 31 cases in the winter of 1909–10, which all came from Portland, Ore., along the lines of railroad and along the Columbia River. There seemed to be no definite foci, and no definite method of spread, except as above stated, of all cases appearing along the lines of travel. All cases were in children under seven years of age.

In the Massachusetts State Board of Health report (65) for 1910, Lovett and Sheppard report the occurrence of the disease for 1910 in Massachusetts. They state that the disease was nearly as prevalent in 1910 as in the previous year, and affected 153 cities and towns instead of 136, as in 1909. A large epidemic center existed at Springfield with 148 cases in the city and a large number in the surrounding towns. Another center existed in Fall River, with 89 cases in the city and others in neighboring towns. The disease occurred in all classes of the community, and it existed under all conditions of sewage disposal and with all kinds of water supply. They state that their researches in the last two years failed to show an excessive amount of dust in the affected localities. Forty-two cases out of 200 giving histories of contagion from others they believe to be of importance. They stated that no definite information as to any one factor was to be found in the antecedents of the attack, since bathing, falls, exposure to heat, overexertion, etc., are

common occurrences in children of the affected ages in the summer season. The distribution of the paralysis was determined by a skilled investigator in a group of cases carefully studied, and it is of importance to note that under those conditions the comparatively frequent involvement of the back, abdomen, neck and face is a matter often overlooked. The percentage of total recoveries from the paralysis within a period of six months and less after the attack was 13.5 per cent. in 200 cases, as against 16.7 per cent. in a similar class of cases in 1909.

Collins (66) makes a plea in this article for the necessity of establishing quarantine in this disease which is now generally considered contagious. He also reports several epidemics, about 20, which have occurred since 1907.

Lovett (81) reports the prevalence of the disease in the United States and Canada in 1910. He reports a total of about 8,700 cases, not counting Canada or such dependencies as Hawaii, and making no account of States reporting "a few," etc. In 1910 the disease was reportable by law in 23 States. In Canada the disease appeared most extensively in the Province of Ontario, with 179 cases, with 75 in British Columbia and 11 in Alberta. New Brunswick reported a few cases, but no deaths, and there were cases in Quebec and Nova Scotia with one in Newfoundland. There was one case in a white laborer in the Canal Zone and an epidemic of 33 cases in Hawaii. In April, 1911, an epidemic of 20 or more cases appeared in Louisiana. He states that investigations of epidemics will show that the disease will be found in scattered foci, with cases radiating from these foci. It will reach its height in the late summer; it will follow lines of travel; it will affect children in the first dentition period; and the mortality rate will be from 3 to 15 per cent.

Dixon and Karsner (83) state that in 1907 some 200 cases developed in the northwestern part of Pennsylvania. As usual the height of this epidemic was reached by the latter part of September, and was stamped out by the latter part of November. A small outbreak of the disease appeared in July and August in 1908. Sporadic cases only occurred in 1909. During the summer of 1910 an epidemic of considerable proportions developed in widely separated sections of the State, reaching a total of 1,076 cases before the winter months. An intensive study of 773 cases showed that 59 gave a distinct history of exposure to previous cases; 437 were males and 336 were females. But 3 cases occurred among colored people. In 44 instances secondary cases developed in the household. One hundred and forty-two cases (18.4 per cent.) recovered without paralysis, and 294 (71.6 per cent.) recovered with considerable residual paralysis. Their figures show that the danger of

transmission to another member of the same household is not great. In 289 instances children slept in the same bedroom, and only 24 contracted the disease. In 9 instances the disease developed in children after having visited communities where the disease was known to be prevalent.

O'Reilly (67) states that there were 309 cases of infantile paralysis in the State of Missouri in 1910, and that there were 326 cases in the five years preceding Jan. 1, 1910. These cases were scattered throughout the State, the greatest number occurring, however, through the northwestern and western parts. A rather extensive epidemic of infantile paralysis prevailed throughout this section of Missouri, apparently bearing a relation to the epidemics occurring in Iowa, Nebraska and Kansas at the same time. From 1905 through 1910, 81 deaths resulted from infantile paralysis in Missouri, and of this number 33 occurred in 1910. Of these, 12 were in Kansas City and 6 in St. Louis. One physician reported that a number of chickens suffered from paralysis shortly after one of the children of the family had been attacked by poliomyelitis, and another reported a similar condition as having occurred among sheep. The disease was epidemic only in Kansas City. In St. Louis it was sporadic. The author also speaks of the 604 cases occurring in Iowa up to Jan. 1, 1911, the epidemic of 1909 in Nebraska, the 189 cases in Kansas in 1910, and the 5 cases in Oklahoma up to Sept. 1, 1910, and 15 cases reported in Illinois. All these States are contiguous to Missouri.

Gundrum (68) states that poliomyelitis has existed in California for a good many years, appearing sporadically and in occasional epidemics. The first case which he was able to get accurate information about occurred in Eureka, Humboldt County, in 1875. The case was a typical one and was followed by paralysis of the legs. Another child was said to have had the same disease at about the same time. He could find the reports of no other cases between 1875 and 1896. In 1897 Sherman (69) reported 8 cases which occurred in the State in the summer of 1896; 7 of these were in San Francisco and one in Napa. Newmark (70) reported 4 cases which occurred in Merced County in the summer of 1898. Woods (71) described an epidemic of "about 50 cases," which occurred in and about San Francisco in 1901, with a recurrence of several cases in 1902. The most extensive epidemic the State had had occurred in the summer of 1910, which was reported to the State authorities by Fleischner (72). The epidemic did not differ in its clinical aspects from other epidemics. One hundred and twenty-five cases were collected, — 120 in 1910 and 5 before October, 1911;

75 per cent. occurred from May to September, 1910. The number of males attacked was double the number of females; 73 per cent. of the cases were under three years of age. In 88 per cent. the onset was sudden and in 12 per cent. was slow; 67 per cent. showed the classical type, and about 25 per cent. showed the meningitic type, with retraction of the head, and the others were not classified on account of imperfection in the reports. Of the cases of paralysis, 23 per cent. occurred in the upper, 73 per cent. in the lower, extremities, and the remaining 4 per cent. were cases in which the trunk and respiratory muscles were involved, causing death. There were 7 deaths, giving a mortality of 7 per cent. Eighty-two per cent. of the cases began with fever, 54 per cent. with gastro-intestinal disturbances. In regard to the contagion, the author was able to find a history of exposure in 11 cases only, or 9 per cent. He states that there were no secondary cases in adults, and but 5 demonstrable secondary cases in children. The disease appeared at widely separated points at about the same time, without any definite geographical order. It was of interest to note that in the cold weather the disease invaded the southern part of the State, which had been previously exempt from infection. All of the towns but one were on railroad lines. The (84) number of cases of infantile paralysis was not nearly as large in 1911 as in 1910, yet the disease was widely distributed throughout the United States. The number reported in 1910 was 5,861, with 950 deaths, and in 1911, 1,931 cases with 440 deaths, according to the public health reports. The report compiled by the Bureau of the Census from the registration area shows the number of deaths for 1910 as 1,459. The data, however, are incomplete, and therefore the number of cases was undoubtedly larger than these figures indicate. These figures bear out our previous knowledge that the disease has alternate active and more or less quiescent periods, owing to lack of susceptible individuals or to attenuation of the infection.

Brues and Sheppard (85) investigated the possible etiological relations of biting insects to the spread of infantile paralysis during August, September and October of 1911 in certain Massachusetts cities and towns, to see what evidence could be adduced to support the theory of infection from insects. They conclude that many facts connected with the distribution of cases and the spread of epidemics of this disease, together with the histories of insect bites, suggest at least that the disease may be insect-borne. They state that the common stable fly may be responsible to a certain extent for the spread of the disease. They also suggest that if the disease should prove to be common to any species of domestic animals, as is now strongly suspected, a secondary

connection of ticks in spreading the disease among such animals seems probable.

Langer (135) reports a study of poliomyelitis in the schools of Steiermark. He believes that schools are a source of infection, and that all possible measures should be taken to prevent the spread of the disease in this manner. He gives detailed reports from 21 schools. He states that he had 60 cases during the school year and 37 during vacation. Where there were several cases in different classes in the same school, twelve times there were 2 cases in the same class, twice there were 3 cases and once 5 cases. In 6 schools the patients were seat mates, and in 10 classes only children of the same sex were affected. In the majority of cases it was found that the children went to and from school together and were closely associated otherwise. At the time of the school epidemics there were also cases among outside children in the neighborhood of the school.

Bruno (74) relates that recently two young children developed infantile paralysis on a poultry farm near Heidelberg, following the receipt of a consignment of ducks from a distance. One duck was found dead on the seventh day after arrival. Seven days later another duck seemed to be paralyzed, and it was killed and eaten. Three weeks after the arrival of the ducks two others developed the same symptoms of paralysis and a week later a third, but all recovered. None of the 47 geese and chickens showed any signs of the disease. The two children developed symptoms on the thirty-ninth day after the arrival of the ducks. The entire family complained of feeling ill for several days with fever and gastrointestinal disturbances. Bruno also reports a recent case of a child with epidemic infantile paralysis on a farm where a cow had just died with symptoms indicating paralysis, and a paralyzed hen was found a little later. On another farm a goat died with symptoms of paralysis, and ten days later an isolated case of infantile paralysis developed in the farmer's family. He states that at present Heidelberg seems to be the center of a widespread epidemic of infantile paralysis, although the cases are not very numerous.

Roth (75) reports an epidemic of 6 cases which involved 5 small villages, separated from one another by not less than 2½ miles. The individuals involved were never in direct communication with one another, nor was there any traceable evidence of the intervention of any possible carrier. They all occurred within a little more than a month in the summer. The one common feature among several cases lay in environment, all cases residing in the immediate vicinity of barns or stables where the fly *Stomoxys calcitrans* was very abundant.

Roth believes that in view of the findings of Rosenau, which have been confirmed by Anderson and Frost, this insect was the means of the spread of the disease. Jubb, quoted by Roth, reported an epidemic of 8 cases in an urban locality where there had been no previous cases. Jubb believes the first case originated from a donkey, which was brought from another region. It seemed possible to trace all the succeeding cases to direct or indirect contact with the first patient, or with subsequent cases. It is interesting to note, however, that after the first case appeared there was an interval of a year before the other patient became infected. This condition, it was believed, was due to the fact that the brother of the first patient harbored the virus and became a carrier, not being affected by the disease himself.

The Ohio State Board of Health (76) in its January, 1914, bulletin has the first installment of a rather extensive review of poliomyelitis, including a study of the disease as it has occurred in Ohio, also a study of the habits and anatomy of the stable fly, and also reports a number of transmission experiments by means of the stable fly on monkeys. They report a number of sporadic cases in Ohio in 1910, which cases later led to an epidemic. The disease was made a reportable one in Ohio in 1910. It became epidemic in 1911. Seventy-six deaths occurred in Ohio in 1910 from poliomyelitis. In 1911, 142 deaths occurred from this disease. In 1911 Cleveland and Cincinnati, the two largest cities of the State, experienced epidemics of poliomyelitis. The epidemic in Cincinnati was preceded by an epidemic in Covington, Ky. There were 103 cases with 41 deaths, the greatest number of cases occurring in October and November. Ninety to 95 per cent. of the cases were under six years of age, with a large majority between one and two years of age. In Cleveland 55 cases were reported in 1911, the first appearing on January 4. There were 15 deaths. The disease remained sporadic until September 19, when 5 cases developed, and after that it assumed epidemic proportions.

In 1912, the disease began to appear in January and prevailed in sporadic form until June, when an increase in number took place. Three hundred and fifty-four cases were reported. Cases were reported from 54 of the 88 counties of Ohio. A tabulation of the cases by seasonal occurrence shows that the largest number of cases occurred during the months of June, July, August, September and October. More cases were reported from cities than from rural districts. There seemed to be no relation to the incidence of the disease and the dust, sanitary arrangements, previous health, trauma, etc. The greater number of cases occurred among the so-called middle class of people. There was

no coincident paralysis among domestic animals. There seemed to have been a possibility of spread by contagion and by contact in about 18 cases. No single school was found to be a focus of infection.

Flexner, Clark and Amoss (138) state that they do not possess a generally acceptable theory to account for the epidemic waves of disease. What is required is an adequate explanation of the initial rise, persistence and the final fall of the wave as represented by the varying number of the affected. They state that the subject has not been rendered essentially more comprehensible by the discovery of the healthy and chronic carriers of infectious micro-organisms, or by the more ready detection of so-called abortive cases of infection. Indeed, these discoveries only add to the perplexity, since they prove that potentially infective micro-organisms capable of starting epidemics are more frequently present in our surroundings than has hitherto been supposed. They state that a factor which has not up to the present been sufficiently considered is that of variations among the micro-organisms themselves that may be directly responsible for the production of epidemics. That micro-organisms, along with all living things, tend to vary in their biological properties has long been known, but it is only recently that these variations have been recognized as constituting mutations. Not a few pathogenic bacteria may be changed profoundly in virulence by animal passages. Most often with the effect of intensification, but not infrequently with the contrary effect, the modifications in virulence appear at all times quickly, or even suddenly, and at another, develop gradually. There is no doubt that under many natural conditions the passage of infectious micro-organisms rapidly from animal to animal, or person to person, leads to great enhancement of virulence. There exists experimental foundation for the belief that during the rise of epidemics the microbic causes are more virulent than during its fall. At the onset the virus of human poliomyelitis possesses relatively weak pathogenic action for monkeys. By means of a few passages the infective power rises, and soon a maximum is reached which endures several years. Ultimately the infective power falls off and soon becomes greatly diminished, so that finally the power is no greater than at the onset. There exists another fact inherent in all epidemics, namely, the varying number of susceptible persons who fall victims to the prevailing disease. In the light of this presentation the part played by sporadic and abortive cases becomes more comprehensible. Such infected persons or animals may be considered as carrying specific micro-organisms lacking high virulence for their respective kind. We then can begin to see how the conversion, through favoring

causes, of the micro-organisms often into others of high virulence may be the signal for the appearance of epidemics, arising almost simultaneously in separated and even remote places when the conditions are similar; just as, on the other hand, the immediate transportation of already elevated micro-organisms from a place in which an epidemic is already prevailing to new places may start a similar severe outbreak there. They state that a strain of poliomyelitis virus was propagated in monkeys for four years, during which time it displayed three distinct phases of virulence. The several phases covered different periods of time. At the outset the virulence was low, but by animal passages it quickly rose to a maximum, which was maintained for about three years, when, without known changes in the external conditions, a diminution set in, and increased until at the expiration of a few months the degree of virulence about equalled that present at the beginning of the passages in the monkeys. The cycle of changes in virulence is correlated with the wave-like fluctuation in epidemics of disease which also consist of a rise, temporary maximum, and fall in the number of cases prevailing.

Lust and Rosenburg (173) report 71 cases occurring between March and December, 1913, in Heidelberg. This is the first time this disease has been epidemic in this region; only 4 of the patients were over four years of age. The cases were scattered over a wide region, 32 coming from different places where only 1 case had been observed; nine places where there had been 2 cases; one place with 4 cases and one with 3. There was found, on investigation, to have been some co-existing and preceding paralysis current among the hens, squirrels and two hares.

Pierson (174) reports an epidemic of poliomyelitis which occurred in the summer of 1913 among the Indians in Central Alaska, on the Yukon River. There were 4 deaths, and residual paralysis in 5 cases. In all there were 30 cases. The epidemic among the human subjects was preceded by an epidemic of "distemper" among the dogs. The symptoms manifested by the dogs were the same as those shown by the human subjects. One woman had visited the camp where the dogs were sick, and shortly after her return she was attacked by poliomyelitis and died. He states that in this epidemic there was every opportunity for the germs to be carried by direct contact, on account of the close association of the Indians and dogs. There was ample chance, also, for the infection to be carried by flies. The disease has been much more prevalent where flies were more abundant. All cases were contracted at or near the fish camp where there were many flies. Other Indians who did not live near the fish camp did not have the disease.

A great many dogs owned by white persons had distemper, but none of the white persons had the disease because they kept the dogs chained away from the dwellings and had few flies about, and had their houses screened. There was no study of the virus in these cases, nor any experimental transmission researches. Many of the persons infected were infested with vermin, and the disease may have been carried by them.

This concludes the study of the epidemiology. It will be noted that the disease occurs in epidemics, is probably contagious, is controlled by quarantine, and can be carried probably by a third person, as noted in the Nebraska report of Shidler (48). The classical type is the predominant one, but meningeal and abortive cases are common. In epidemics there is a relatively high mortality, from 7 to 20 per cent., due to respiratory involvement. The onset is sudden, generally associated with fever, gastrointestinal disturbance and generally constipation. When the legs are affected there may be retention of urine. About 25 per cent. of the cases make complete recovery, but no one can tell which case is the one which is going to make such a recovery. Such recovery usually occurs rapidly, that is, in the course of the first six or eight weeks. The disease is one largely of the summer months, and attacks usually children between two and three years of age, although adults and elderly persons may become affected.

2. Experimental Etiology, Modes of Transmission, Bacteriology and Pathology.

In the study of the etiology, modes of transmission, bacteriology and pathology of anterior poliomyelitis there are many points of interest, which have been worked up to a fairly final stage and have given us certain definite facts to use in dealing with this disease. A review of the great amount of literature which has been rapidly accumulating during the past seven years, beginning with the studies of Harbitz and Scheel in Norway in 1907, has been undertaken. In this section I will take up each paper of interest and value which has been published since then so as to offer to the reader a general survey of the field so far covered, which will give him an adequate idea of the subject from these various points of view, and also an idea as to the great amount and value of the research work which has been done in the hope of finding a definite answer to the various so far unsolved problems to be met with in this increasingly prevalent disease.

Harbitz and Scheel (77) in an anatomical investigation of the nervous system of 19 cases of acute poliomyelitis, found in the spinal cord a

diffuse infiltrating inflammatory process closely related to the blood vessels, and chiefly in the gray matter, and within this, chiefly in the anterior horn. Generally the inflammation extended along the whole length of the cord, but was most intense in the cervical and lumbar enlargements. The degeneration of the ganglion cells was generally very marked, and extended rapidly over large areas of the cord. There were numerous "heaps" of leucocytes corresponding to destroyed ganglion cells. It was remarked that the inflammation was generally more than was to be expected from the clinical symptoms, and often took on a hemorrhagic character. Microscopic evidences tended to show that the pia mater was involved first, and that the inflammation then extended to the cerebrospinal fluid and the cord. The inflammation reached its greatest intensity in the anterior gray horns, because they are supplied by the most numerous and largest blood vessels. The symptoms of meningeal irritation seen early in many cases also tends to show that the meninges are involved first. They also found more or less extensive inflammation in the brain substance, most pronounced, however, in the medulla oblongata and pons.

They speak of Geirsvold as having found a diplococcus in the spinal fluid of 12 cases. He inoculated animals with the germ and produced paralysis and death. Up to this time this diplococcus has been accepted as the causative agent in this disease.

Harbitz and Scheel (86) speak of certain micrococci which they found in 3 of the cases they had previously reported (77). These bacteria were demonstrated in the spinal fluid, and proved to be bean-shaped diplococci, which grew in from two to six days on artificial cultures. They speak of a number of other men who have found similar diplococci in other cases of poliomyelitis. The cocci proved virulent for animals in that they caused atrophy, paralysis, emaciation and death.

Pasteur, Foulerton and MacCormac (134) report a case of non-fatal acute poliomyelitis, in which they were able to identify in the spinal fluid withdrawn during life a micrococcus which produced on injection, into a series of rabbits, symptoms resembling the disease in human subjects. The same micrococcus was identified in the spinal fluid of the experimental animal, although they were unable to produce it by culture.

Landsteiner and Popper (87) claim that they have transmitted the disease to monkeys. They seem to have been the first to have attempted to communicate the disease to animals by direct inoculation. They used parts of a spinal cord of a boy who died of poliomyelitis, the inoculations of this material emulsified in salt solution being made into

the peritoneal cavity of monkeys, rabbits, guinea pigs and mice. Only two of the monkeys showed any effects of the inoculation, one becoming severely ill on the sixth day and dying two days later, and the other becoming completely paralyzed in the hind legs about seventeen days after the inoculation. Pathologically, both these animals showed the typical lesions of poliomyelitis. They were unable, however, to propagate the disease experimentally beyond the first generation, and they suggested that the virus of poliomyelitis probably is not demonstrable by the usual methods used to study bacteria, but might be a virus of protozoön nature.

Flexner and Lewis (88) tried to transmit the disease to lower animals first in 1907, at which time cerebrospinal fluid, obtained by lumbar puncture, was introduced into the spinal canal and peritoneal cavity in monkeys and other animals. Their results were negative. In 1909 they introduced, through a trephine opening in the skull of monkeys, portions of a spinal cord from a case of poliomyelitis six hours after death of the patient. The injected material consisted at first of emulsions in salt solution of the spinal cord from the children, and later of emulsions of the spinal cord of monkeys who developed paralysis. By this means they proved that the disease could be transmitted through an indefinite number of monkeys. A delay of unsuccessful inoculation could be converted into a successful one by reinoculation with an active virus. They state that since poliomyelitis has long been supposed to be an infectious disease, and that its successive transfer by an active agent has been accomplished, any further doubt as to its infectious origin can hardly longer be maintained.

Robertson (44) reports the pathological findings in three fatal cases in the Minnesota epidemic of 1908, and states that microscopically the lesions in every case have resembled each other, namely, congestion of the vessels, especially those leading to the anterior horns; perivascular infiltration of polymorphonuclear and mononuclear cells; infiltration of pia and nerves running from the anterior horns; necrosis of the gray matter of the anterior horns, especially the ganglion cells, with diffuse infiltration of polymorphonuclear and mononuclear cells; hemorrhages into the gray matter of the anterior horns; thrombosis of vessels in region of hemorrhages; dilated lymph channels; and occasional infiltration along the lines of vessels extending to base of brain and also posterior horns of the cord.

Flexner and Lewis (89), in a second article dealing with the nature of the virus of epidemic poliomyelitis, conclude that in view of the negative result of bacteriological and histological examinations the infecting

agent in this disease belongs to the class of the minute and filterable viruses that so far have not been demonstrated with certainty by the microscope. They also state that the virus of poliomyelitis can be transferred to the central nervous system by way of the subcutaneous tissues in monkeys, and also that the two viruses have each been passed through six series of animals.

In another paper Flexner and Lewis (90) point out how they succeeded in detecting the virus of poliomyelitis in the nasal and pharyngeal mucosa of the monkey, from which they concluded that one path of elimination of the virus was by way of the nasal mucosa, through its lymphatic connection with the pia arachnoid of the brain. They showed by experiment that by inoculating a monkey through a scarified nasal and pharyngeal mucosa infection resulted. They also proved by experiment that the virus of poliomyelitis was contained not only in the central nervous system but in the mesenteric lymph glands, which suggests that it may be present, also, in other organs in human subjects of the disease.

Flexner and Lewis (91) in a further contribution show that the spinal fluid withdrawn on the third day, which is several days in advance of the appearance of the paralysis, contains the virus, and when injected intracerebrally may set up a paralysis in another monkey. They state that the virus is quickly destroyed by a dilution of perhydrol containing 1 per cent. of hydrogen peroxide.

Weinicke (92) reports his experimental findings following an investigation with monkeys and rabbits. He found that the incubation period was usually from eight to fourteen days, but varied from three to forty-one; in using an attenuated virus, a considerable per cent. of the inoculated animals remained well instead of dying, and that the incubation period was lengthened. In a second paper (94) it is stated that the virus in man and infected animals is found not only in the central nervous system, but also in the blood and parenchymatous organs, and is also found in cadavers.

Levaditi (95) observed the constant presence of a great number of extremely minute corpuscles stained deep red by Löffler's method, following the incubation of an active Berkefeld filtrate in bouillon for fifteen days. Monkeys inoculated by this filtrate following incubation became paralyzed.

Landsteiner and Levaditi (96) undertook some experiments to determine the transmissibility of the disease in a series from monkey to monkey. They were able to infect a chimpanzee by injecting 5 cubic centimeters of an emulsion of the cord of a baby which had died of the disease into its peritoneal cavity. Following the death of the chimpanzee they were able to transmit the disease to monkeys by injecting

an emulsion of the chimpanzee's cord into their peritoneal cavity and brains. The period of incubation from injections to first symptoms is from seven to eleven days, — the longest period twenty days. The incubation period is prolonged by the use of small amounts of the virus. The pathological lesions were the same as those found in human cases. They feel that the toxins act directly on the nerve cells, and that the degeneration of the neurons is not dependent on vascular lesions. The microbe invades the nervous system through the perivascular lymph spaces, and when it reaches the gray matter attacks the nerve cells. They also state that emulsion taken from animals more than a few days after the acute stage of the disease does not carry the infective agent. One attack produces immunity in animals, and the serum of cured animals has destructive powers over the virus of the disease which is most suggestive.

Strauss (97) tried in 10 cases to inoculate monkeys with infantile paralysis by injecting the filtrate of nasal swabbings from patients ill with the disease into these monkeys. He was wholly unsuccessful.

Knopfelmacher (98) attempted to carry over to a second generation the disease with which he had inoculated a monkey by the peritoneal route, by means of the spinal cord of a child which had died of poliomyelitis. He was unsuccessful.

Leiner and Weisner (99) conducted a research on acute poliomyelitis, and state that the intracranial method of inoculation in apes offers more reliable results than the intraperitoneal method. The various micro-organisms which have been described by different authors in the past, as the cause of the disease, could not be identified, and they consider that such micro-organisms must be looked upon as accidental findings.

This view has been concurred in by Duval (100) of New Orleans, who states that there is a complete absence of "uniformity in the bacteriological examinations of the spinal fluid in cases of poliomyelitis thus far recorded, and the results are without the slightest value." In connection with this statement we may turn to the paper of Strauss and Huntoon (101), based on the careful experimental investigation of one fatal case. Cultures from the cord in this case on agar showed a bacillus which, injected intraperitoneally into a monkey, produced acute poliomyelitis; but this disease could not be transferred to other monkeys. The authors think that the cerebrospinal fluid does not contain the virus in an infective state, and that the bacterial findings in this fluid are either contaminations or secondary invaders.

Krause and Weinicke (102) made examinations from the blood, spinal fluid, tonsils, stools and urines of living cases. Cultural experi-

ments and injections into animals failed to reveal the specific cause of the disease. They were able, however, to infect rabbits by the intraperitoneal route; after long periods of good health the rabbits showed changes in the nervous system and died.

Russell (103) thinks that the disease is a general infection which affects more particularly the spinal nervous system and meninges, and not alone the gray matter supplied by the anterior spinal artery. He thinks that we get sensory changes as well, which are not detected because we generally are dealing with children in whom the testing of sensibility, when only a relative impairment is expected, is impossible.

Flexner and Lewis (104) note that the disease can be transmitted readily from monkey to monkey by using the intracerebral route, possibly through an indefinite series; and also that it is possible to transmit the disease successively by means of inoculating the sciatic nerve, the circulation, the peritoneum and subcuticular tissues.

They also have shown that the virus was filterable through a Berkefeld filter and withstood glycineration. They have produced the disease by injecting an emulsion of regional glands, which communicated with a nodule caused by a subcutaneous injection of the virus. In regard to the resistance of the virus they state that the spinal cord from a human case retained its virulence after being kept frozen at 2° to 4° C for forty days. They state that these experiments have a distinct bearing on the epidemiology of the disease, and indicate that the cessation of the cases which occurs with the onset of cold weather does not depend on the destruction of the virus. Experimental evidence tends to show that an attack of poliomyelitis affords immunity to reinfection.

Lucas (105) has examined the blood of monkeys before and after inoculation, and found that in the acute stages of the disease there was a moderate to a constant lymphocytosis and an eosinophilia. Parallel with this lymphocytosis there was a marked and constant leukopenia. This drop in the white count lasted fairly consistently with the acute state, disappearing about the time that the hyperæsthesia or other manifestations of the acute stage disappeared. The count did not change during the incubation period, but did drop occasionally during the prodromal stage.

The spinal fluid also showed even more marked characteristics during the various stages. Before inoculation a dry tap was the rule. During incubation there was a marked increase in the spinal fluid, and the cells were markedly increased. These cells were mainly the large mononuclear type. In the prodromal stage there is even a more marked increase in these same cells.

In this prodromal stage polynuclears are still present as high even as

60 per cent. In the early acute stage the increase is very marked, but cells of the lymphocytic variety predominate. As the cells decrease in number the polynuclears begin to return, and at the end of a week or ten days there are very few cells present. In an examination of the spinal fluid of four children suffering from the disease, similar findings were observed.

Gay and Lucas (106) also tested the serum of inoculated monkeys for antibodies unsuccessfully, and also had negative findings for antigens in the spinal fluids of monkeys and human beings at various stages of the disease.

Flexner and Lewis (108), in a note on a mode of spontaneous infection, state that the experimental results show that a path of elimination of the virus of poliomyelitis is by way of the nasopharyngeal mucosa, and that the infection may also traverse the same path. They believe, therefore, that it is desirable to destroy the secretions of the nasal and buccal cavities as a matter of prophylaxis. They state that Levaditi and Landsteiner (96) found the salivary glands contained the virus.

Rosenau (65) reports to the Massachusetts State Board of Health his attempts to transmit the disease to monkeys by inoculation with the nasal, pharyngeal and buccal secretions of eighteen human cases. He reports in detail the method of collecting the material by washing the nose and throat with salt solution, and also the detail of each case with the report of the inoculated monkey. He was unsuccessful in producing paralysis in any of the monkeys, and believes that some of the factors in the failure were possible attenuation of the virus and dilution of the virus from such large amounts of salt solution as were necessary to collect the material.

Skoog (109) reports the results of some of his experiments for the Kansas State Board of Health, at the University of Kansas. A number of animals in the fall of 1909 were used, including chickens, guinea-pigs, rabbits and three monkeys. Cerebrospinal fluid and blood from infected cases were introduced into these animals, all intraperitoneally, excepting for one intraspinal injection in a monkey. The results were all negative. In the summer of 1910 he experimented with some other monkeys, and was able to produce the disease by inoculation from human and monkey virus, in two monkeys, by using the intracranial route. Other inoculations failed.

Osgood and Lucas (110) report a most interesting and suggestive series of transmission experiments, having used the nasopharyngeal mucosa of monkeys, recovered from the acute stage of poliomyelitis for purposes of inoculation.

Beginning with a well monkey they inoculated it with an active virus

intracranially. He became completely paralyzed in nine days, and died five months later. The nasopharyngeal mucous membrane was removed, ground up in sterile salt solution, and passed through a Berkefeld filter. Monkey No. 2 was now injected intracerebrally with 7 cubic centimeters of this filtrate. He died in five days, showing prodromal symptoms of the disease, and an autopsy showed the characteristic lesions of poliomyelitis. Two cubic centimeters of the same filtrate were injected in the same way into monkey No. 3, but he failed to show any symptoms and remained well.

Monkey No. 4 received by the intracerebral route 6 cubic centimeters of the emulsified cord and brain of monkey No. 2, which had died with paralysis. It became paralyzed in seven days, and was sacrificed, the autopsy showing a typical poliomyelitis. Monkey No. 5 was given 6 cubic centimeters of the emulsion from the cord of monkey No. 4. It became paralyzed in four days and was killed. The autopsy showed typical lesions. No. 6 was given 5 cubic centimeters of the filtrate of the cord of No. 1, which had been preserved in equal parts of glycerin and salt solution, but it developed no symptoms.

No. 7 was given 2 cubic centimeters of a 5 per cent. emulsion in salt solution which had been obtained from Dr. Flexner. Paralysis appeared in eleven days. It was killed about five weeks later, the nasopharyngeal mucosa was obtained, ground up and passed through a Berkefeld filter, and 10 cubic centimeters of this were injected into monkey No. 8, which died two days later of respiratory failure. The nasopharyngeal mucosa was obtained and treated in the usual way, and 7 cubic centimeters were injected into monkey No. 9, which developed paralysis two weeks later and died the next day of respiratory failure, showing typical lesions of poliomyelitis. No. 10 was given 5 cubic centimeters of the filtrate from the cord of monkey No. 8, and in two days there was complete paralysis, the autopsy showing the usual lesions.

Their experiments showed that they were able to transmit from monkey to monkey a typical poliomyelitis from the filtrate of the nasopharyngeal mucosa of two monkeys six weeks and five and one-half months, respectively, after the acute stage of the disease had passed. They also found it impossible to transmit the disease by intracerebral inoculations of the cord and brain of these recovered subjects, or from the filtrates of the nasopharyngeal mucosa of a monkey in good health, which had received a previous intracerebral inoculation of an active virus, and had lived in close contact with monkeys in the acute stage of the disease. Their observations show that the virus can persist in a viable and infectious state in the nasopharyngeal mucous membrane in the monkey for several

months after the acute period of paralysis has passed, and for a far greater time than it survives in the central nervous system.

Anderson and Frost (111) examined blood specimens from a number of abortive cases of poliomyelitis, and inoculated monkeys with equal parts of the blood serum and active virus. They believe from their results that normal human serum may have a germicidal action on the virus of poliomyelitis. No difference was shown between the normal serum of adults and of children, in regard to their action on the virus. The serum of patients who have had the disease and recovered, exhibits a germicidal action on the virus considerably greater than that exhibited by normal serum.

Neustaedter and Thro (112) collected the dust of rooms wherein cases of poliomyelitis had occurred, and prepared extracts from these sweepings, which they injected subcutaneously, intraspinally and intracerebrally into monkeys. The sweepings were obtained from cases of from six months' to three days' duration after the onset of the paralysis. They succeeded in producing paralysis in a monkey by an intraspinal injection, by using the sweepings of a room which had been occupied by a child with acute infantile paralysis for two days. The dust was macerated overnight with sterile water and then filtered through paper and then through a special Bongé filter. This filtrate was then injected with the above result. The paralysis occurred six days after the injection. The autopsy on the monkey showed typical lesions of poliomyelitis. The authors believe that they have proved that acute poliomyelitis is both infectious and contagious; that it is propagated by dust; and that the nasopharynx must be the port of entry.

Flexner and Clark (114), in their eleventh note state that the virus has been found to be present in the tonsils, in that they were able to produce the disease experimentally in monkeys by injecting emulsions intracerebrally and intraperitoneally. They state that the virus has not been found in the blood of human beings, and it has been found in the blood of monkeys at the height of the disease only when large quantities are withdrawn and injected intravenously into a healthy monkey. The cerebrospinal fluid fails to show the virus at the onset of the paralysis in human beings and monkeys. It has been detected, however, in the spinal fluid of monkeys three or four days after an intracerebral injection and during the incubation stage. They state that the human strain of the virus not only infects monkeys less readily than do the modified or monkey strains, but the experimental disease produced by them is less severe and less fatal.

Lucas and Osgood (115) carried out a series of experiments in order

to determine the protective value of certain specific sera and vaccines against the virus of poliomyelitis for the Massachusetts State Board of Health in 1911. They concluded that in the monkey a partial and perhaps complete immunity to the infection of dysentery, streptococcus pyogenes, typhoid fever, gonorrhœa, pertussis, and staphylococcus pyogenes affords no demonstrable protection against the virus of poliomyelitis. They state that Landsteiner and Levaditi (Annales de L'Institut Pasteur, November, 1911) found that specific inoculations carried out with the virus of rabies, a disease much more closely allied to poliomyelitis than any of the infections above detailed, produced in monkeys no immunizing effects whatever.

Pettersson (116) states that large amounts of the virus of poliomyelitis were found by him in secretions from the mucous membrane of the nose, pharynx, trachea and intestines. He was able to demonstrate the presence of the virus by inoculating monkeys. He stated, also, that there is nothing against the hypothesis that epidemics are spread by direct or indirect transmission of the virus from person to person. However, there is a good deal against the supposition of the transmission of the virus by insects to such a degree as to cause epidemics.

Flexner, Clark and Dochez (117) experimented on a monkey for the purpose of determining the viability of the virus of poliomyelitis in the stomach and intestines, and concluded that since the virus occurs in the nasal and buccal mucous in humans, it is inevitably swallowed. The virus survives the action of the gastric juice and intestinal secretions, and persists for a time in these organs. In human beings it leaves the body in part with the intestinal discharges, which are consequently potential sources of infection.

Rosenau and Brues (118) exposed monkeys in all stages of the disease to the bites of *Stomoxys calcitrans,* or stable fly. The monkeys had been infected in the usual way by injections. After the flies had had ample opportunity to bite these infected monkeys during the various stages of the disease, including the inoculation period, healthy monkeys were then exposed to the bites of these same flies. Out of 12 healthy monkeys 6 had indications of the disease, 3 in virulent form, resulting in death, and 3 with partial paralysis, but recovered. No conclusions are drawn as to the significance of these experiments.

Flexner (119) recapitulates in regard to the present knowledge of the disease. He states, besides, that the virus has not been found as yet in the spleen, kidneys, liver or bone marrow. He states that it escapes from the body by the nose, throat and intestines. He states that it has been established that the virus passes readily from the intact,

or practically intact, mucous membrane of the nose to the central nervous system, and that this membrane, next to the direct intracerebral introduction of the virus, provides the readiest method of successful inoculation. He believes that the clinical evidence is strong in suggesting that human carriers of the poliomyelitis virus exist. The virus has been detected in the secretions of the nose, throat and intestines of persons suffering from abortive or ambulant attacks of poliomyelitis. The unrecognized cases of abortive poliomyelitis play a highly important part in the dissemination of the virus, through which the area of infection is extended.

A similar part has been accorded by clinical observation to the healthy virus carrier. House flies may act as passive contaminators, since the virus survives in these insects. Bedbugs have been infected with the virus, which remained alive in these insects for many days. A filtrate prepared from these bedbugs and injected into monkeys caused them to develop the characteristic paralysis. He believes that the extension of the disease is limited by a high natural indisposition or insusceptibility to infection existing among persons of all ages.

Anderson and Frost (120) exposed three monkeys daily to the bites of several hundred *Stomoxys*, which at the same time were allowed daily to bite two inoculated monkeys. These first three monkeys developed typical poliomyelitis eight, seven and nine days, respectively, from the date of their first exposure. In order to confirm the diagnosis of poliomyelitis in these monkeys, 1 cubic centimeter of an emulsion of the cord from one of them was injected intracerebrally into a healthy monkey, which on the third day following developed a partial paralysis of the foreleg. The next day the animal became completely paralyzed and died. At autopsy histological examination showed the characteristic lesions of poliomyelitis, intense congestion and perivascular infiltration, foci of round cell infiltration here and there in the gray matter, destruction of the cells of the anterior cornu, and small hemorrhages in the anterior and posterior cornu.

One point of especial interest in the results reported by Anderson and Frost is the period elapsing between the first exposure to flies of the infected animal and the development of the disease in the healthy monkeys, the shortest period being seven days, which is shorter than that found by Rosenau (118) in his experiments.

Langhorst (121) states that dogs are numerous in the countries that have suffered most with poliomyelitis; dogs are also more numerous in the country districts where the disease is most prevalent. He reports two cases in detail, one of a man of thirty-five who died of the disease two or three weeks after he had cared for his dog which had paralysis

of the hind legs, and which had licked his hand over an abraded area. He believes that the dog could have inoculated his master with the secretions from his nose and throat. The second case was in a boy of six who developed paralysis about three weeks after having been bitten by a pet dog.

Howard and Clark (122) claim that the domestic fly (*musca domestica*) can carry the virus of poliomyelitis in an active state for several days on the surface of the body, and for several hours in the intestinal tract. Mosquitoes have not in their experiments taken up and maintained in a living state the virus from the spinal cord of infected monkeys. Lice have also failed to do so. The bedbug has, however, maintained the virus in an active state for seven days. House flies cannot be viewed as actual inoculators of the virus, however, since these insects do not bite; but as their power of flight is great, they therefore must be considered as potential passive contaminators, theoretically capable of carrying and depositing the virus at a considerable distance from the original point of infection. Through the ordinary active habits of the fly the virus may be transferred to persons or things with which persons come in close relation, and by their death the flies may through disintegration liberate surviving virus that may become dust.

Flexner, Clark and Fraser (123) describe an instance of the demonstration of the virus of poliomyelitis on the upper respiratory mucous membranes of healthy human adults, — the parents of a child suffering from an acute attack of epidemic poliomyelitis. The patient was taken sick on Oct. 12, 1912, and developed paralysis October 17. On October 28 the mother and father were subjected to a nasopharyngeal irrigation of normal salt solution. This fluid was passed through a Berkefeld filter and injected into the sheath of each sciatic nerve and into the peritoneal cavity of a monkey. Paralysis developed in the monkey on November 12, and the animal was killed the next day, the autopsy showing typical lesions of poliomyelitis. Emulsions of the glycinerated spinal cord of this monkey were injected into each sciatic nerve and into the peritoneal cavity of two monkeys. Six days later one of the animals showed prodromal symptoms, and four days later was killed, the pathological examination showing the typical infiltrative lesions of poliomyelitis. The other monkey became paralyzed sixteen days after the inoculation, and four days later was almost wholly paralyzed and was killed. The autopsy showed the usual characteristic lesions. These experiments offer undoubted evidence of the occurrence of the virus of the disease in the nasopharynx of healthy persons who have been in close contact with an acute case of poliomyelitis, and afford an experimental basis for the

belief, based on clinical observations, of the occurrence of passive human carriers of the infection.

Flexner and Noguchi (124) note the results of their efforts to cultivate the virus of poliomyelitis, using the methods employed by Noguchi in the cultivation of spirochetes, and worked out by him. They state that the virus belongs to the class of filterable organisms, and passes readily through Berkefeld filters, and that up to the present it has not been rendered certainly visible.

The culture mediums consisted of sterile, unfiltered ascetic fluid, or of brain extract to which fragments of sterile rabbit kidney and a layer of paraffin oil have been added, and of these plus 2 per cent. nutrient agar-agar in proportion of one to two.

The first mediums permit of a slow growth not visible to the naked eye, while the second (which are unsuitable for obtaining the initial growth) yield after several days, visible minute colonies clouding the tube. These cultivations are conducted under anaerobic conditions. The minute colonies are composed of globular or globoid bodies. These bodies appear in a variety of arrangements, — single, double, short chains and masses. The cultivated bodies stain a pale reddish violet in Giemsa's solution, and bodies of identical appearance have been demonstrated by Noguchi, in films prepared directly from the nervous tissues. Cultures have been inoculated into monkeys and have caused the typical lesions of poliomyelitis, and from the nervous tissues of these monkeys other animals have been inoculated and the culture recovered in pure culture.

McIntosh and Turnbull (125) succeeded in infecting monkeys with poliomyelitis from two cases of sporadic disease. Both of these strains have been transmitted through a series of monkeys. These are the first recorded successful transmissions of the disease from man to monkeys in England, in which the virus has been obtained from English cases. The authors believed that their previous failure to infect monkeys was due to the fact that the infective agent is rather avirulent, as it is seen in England, where the disease is only slightly epidemic. The monkeys were inoculated intradurally as well as intraperitoneally with emulsions of the cord. The authors suggest that in sporadic cases the virus is feeble, and the occurrence of the disease is due to a hypersusceptibility of the individual attacked, while an epidemic only results when the virus has become exalted either by a series of passages through susceptible individuals, or from some other unknown cause.

Lucas and Osgood (126) report finding the virus of the disease in the nasal secretions of a human carrier four months after the acute

stage of a second attack of poliomyelitis. They state that others have also found the virus in washings from the nasopharynx of parents, attendants and friends, and also that the virus has been found as long as two hundred and four days after the infection in the nasopharyngeal washings. They report the case of a boy who had an attack of poliomyelitis in February, 1910, and a second attack in September, 1912. In November, 1912, nasal washings were obtained with which two monkeys were inoculated, with negative results. Later, January, 1913, more nasal washings were obtained, and two monkeys were inoculated, with negative results. This was repeated later in January, 1913, and the monkey became paralyzed and died; the pathological findings were not typical, but were suggestive.

Jan. 31, 1913, the injection was repeated with a fresh nasal secretion, and the monkey died eight days later, completely paralyzed. Two monkeys were then inoculated, one with a cord emulsion of this last monkey, and one with a cord emulsion of the monkey which died with atypical pathological findings, but which was paralyzed. Both of these monkeys died with typical poliomyelitis, demonstrating, first, the long period during which the virus was harbored in the nasopharynx; secondly, that the boy's sister was infected two years after the original attack; and thirdly, the recovery of the virus from his nasal secretions four months after his second attack, and two years and three months after his first attack. Also that the successful inoculations were due to filtrates from straight nasal secretions and not from washings.

Proescher (127), in a discussion as to the etiology of poliomyelitis and variola, states that the virus of poliomyelitis is constantly found in pure cultures in the central nervous system, and is seldom found in the large internal organs. It belongs to the easily filterable viruses, and passes through the Berkefeld filters V and N, Chamberland, Reichel and Pukall filters. He states that the virus of rabies, poliomyelitis and variola can be made visible with methylene azure carbonate, and that their invisibility is therefore not due to their submicroscopical size, but to their special staining properties. The size of these viruses averages the same. He believes that the term filterability does not mean microscopical invisibility, for other investigators have shown that the smallest visible forms of viruses are about 0.1 micron to 0.2 micron, which border on the line of microscopical visibility. This size corresponds to that of the smallest pores of the filter, through which the micro-organism can pass.

The poliomyelitis virus is the easiest filtered, through all kinds of filters. The variola and rabies viruses are very difficult to filter, especially the rabies virus, which will pass the Berkefeld filter only in small

quantities. He states that the difference in the filterability of these viruses demonstrates clearly that not only the size but also the individual differences in the chemical constitution of the protoplasmic substance play a rôle in the filterability. If the size alone controls the filterability, all these viruses should pass the same filters with the same ease as the poliomyelitis virus. He believes that the staining properties of these viruses may give a key to their chemical constitution, which will satisfactorily explain not only the cause of filtration, but their other peculiar biological properties.

Rosenau (128), in an address to the Massachusetts Medical Society in June, 1913, as to the mode of transmission of poliomyelitis, states that in spite of the large amount of research work done on this problem, the mode of transmission still remains unsolved. Of the two methods, namely, epidemiological field studies and laboratory research, of approaching the problem, the epidemiological evidence is less to be trusted, for he states that sanitarians know well that it has always been necessary to revise the chapter on the epidemiology of a disease as soon as its mode of transference is discovered. The four chief theories of transmission are: —

1. That it is a "contagious" disease, communicated directly from person to person through the secretions from the mouth and nose.
2. That it is an insect-borne disease.
3. That it is conveyed through dust.
4. That it is an alimentary infection, the virus being taken in with food and drink and absorbed from the intestine.

He states that there is evidence from both field and laboratory to support each one of these theories. He then goes on to discuss these theories and the data which have been put forward in support of each, and which are covered in the previous review of literature in this paper.

In concluding he states that in the present state of our knowledge, a definite answer cannot be made to these important queries, *i.e.*, as to its mode of transmission, and that we shall have to await further research before the health officer can combat infantile paralysis with any assurance of success.

Kling (136) reports that he found, in a research on the virus and mode of transmission of infantile paralysis, that 78 per cent. of the monkeys contracted the disease after inoculation with water in which the organs and mucous membranes from poliomyelitis cadavers had been rinsed. The virus was found constantly in the secretions from the nose and throat, and in the intestinal contents of patients sick with acute poliomyelitis, and also in those patients with light abortive cases of the disease, and further, in numbers of healthy contacts. He states

that the virus soon loses its virulence, so that isolation need not be kept up for more than two weeks, although the virus was found in the nasopharynx up to seven months in a few of the cases. He thinks that it is probable that each abortive case is surrounded by a number of carriers, but serious epidemics do not occur, as the persons exposed are not susceptible.

It seems to be the rule that a region where the disease has been epidemic is spared further inroads of the disease later, indicating, maybe, that a large part of the population had become immunized by having had the disease in an attenuated form.

Kling, Wernstedt and Pettersson (129) report that in nine cases of infantile paralysis the secretions from the mouth and intestines were obtained by washings, and monkeys were inoculated with these washings at various intervals. Almost all the monkeys inoculated in the first few weeks died, and only one died after inoculation after seven months. In only one case were the secretions harmless as early as by the end of the first month. There was, however, a steady decrease in the degree of virulence as time elapsed. Inflammatory exudate in the spinal cord was found only in the monkeys inoculated in the first two or three weeks. After that the chief change noted was a degeneration of the nerve cells, which showed diminished virulence of the toxin. They state that it would be manifestly impossible to isolate patients for the several months during which the secretions might be infectious, but if they are isolated for the first few weeks of greatest virulence, the disease will appear only in the milder or abortive forms.

Haywood (137) reports a case in detail of a boy who two weeks previous to the onset of the disease had been bitten on the lip by a dog. Examination of the dog's brain showed the presence of the Negri bodies. Up to the time of the development of the poliomyelitis, the boy had received fifteen injections of the rabies serum. He developed an acute attack of poliomyelitis overnight, involving arms, legs and bladder, from which he partially recovered eventually.

Sawyer and Herms (130) report the failure of their experiments to transmit the virus of poliomyelitis from monkeys suffering from poliomyelitis to healthy monkeys by means of the stable fly. They also failed to produce poliomyelitis in two monkeys inoculated intracerebrally with ground and filtered flies which had fed on the blood of a monkey sick with poliomyelitis. In one instance the flies had just ingested the blood, and in the other they had bitten the infected monkey four and five days before. They also failed to transmit the disease through frequent alternate bitings of sick and well monkeys, followed by daily feedings on healthy monkeys. They were also unable to transfer the

disease to well monkeys by flies which had bitten in rapid alternation well monkeys and a sick monkey painted with his own nasal washings, feces and saliva.

Poliomyelitis had not been produced in a well monkey by stable flies even when they had to drive their proboscides through a layer of highly infectious brain tissue in order to pierce the skin. The same flies did not transmit the disease on subsequent bitings of two other monkeys. These results differ markedly from those obtained by Rosenau and Brues (118) and by Anderson (120).

It is well known (131) that epidemic poliomyelitis does not attack all those who are exposed to the infection. In some cases the attack is so mild as to be recognized only with difficulty. What is the reason for this apparent variation in susceptibility? Kling and Levaditi (96) have observed that the serum of patients who are in the midst of an attack, or who have passed through an attack, destroys or neutralizes the poliomyelitis virus. This has been demonstrated by injecting monkeys with suitable mixtures of virus and serum. Tests of this kind with the serum of children who appeared to be refractory to poliomyelitis, because they remained well while living in the midst of a heavily infected district, gave, in 2 out of 9 cases, complete protection; in 5, partial protection; and in 2, none at all. This condition may be supposed to be the result of an acquired immunity due to a spontaneous protective vaccination or inoculation, in that the virus invading the upper respiratory passage multiplies sufficiently to produce a distinct antigenic effect, but no other appreciable reactions. In order to test the correctness of this theory it would be necessary to repeat the experiments on the protective action of the serum with the serum of individuals who have not been exposed to the virus, and if it should be found that the serum of such individuals is devoid of protective action the theory in question would be greatly strengthened. If, on the other hand, such serum were found to be protective, we should have to conclude that a natural inborn immunity to poliomyelitis exists in certain individuals.

Dubois, Neal and Zingher (132) state that they passed the feces from a typical case of poliomyelitis through a Berkefeld filter and injected the filtrate intraspinally into a monkey, with negative results. They also state that there was a rapid loss of virulence of the brain and cord of the two monkeys, as shown by intraperitoneal injections of two other monkeys, with negative results.

Boudreau, Brain and McCampbell (76) report for the Ohio State Board of Health certain transmission experiments on monkeys of the virus of poliomyelitis by stable flies. They were unable to transmit the

disease in this manner, and also showed that the monkeys bitten by flies which had previously fed on sick monkeys acquired no immunity to infection, in that they were subsequently inoculated with an active virus intraperitoneally, and exhibited typical paralysis and histological lesions. They also attempted to transfer the disease by means of bedbug bites, but were unsuccessful. Fowl ticks were also used, but proved unsatisfactory, in that it appeared impossible for them to digest monkeys' blood.

Flexner (133) says: —

The pathological effects are of two kinds: injury to nerve cells not in the anterior gray matter alone, but in the posterior gray matter of the spinal cord, and in the intervertebral ganglia, medulla and brain, and cellular invasion of the pia-arachnoidal membrane of the spinal cord and medulla that follow the blood vessels into these parts, and pass into the adjacent gray and white matter. The altered vessels permit an escape of albuminous fluid and blood cells into the meshes of the membrane where they mingle with the cerebrospinal liquid, and into spaces in the tissue composing the solid white and gray matter. Sometimes the nerve cells, sometimes the meninges, vessels and supporting tissues suffer most.

When the nerve cells are extensively injured the paralysis is marked; when the meninges are much affected the symptoms are like those of meningitis. The virus of poliomyelitis displays a high affinity for nervous tissues, but it is the wide involvement of the nutritive vascular system in the pathological process that subjects the sensitive nerve cells to so high a degree of injury and destruction.

Flexner, Clark and Amoss (139) state in a contribution to the pathology of epidemic poliomyelitis that the virus of poliomyelitis is neurotropic and localizes, and probably is capable of multiplying in the extramedullary parenchymatous nervous organs. It has been demonstrated by inoculation tests in the intervertebral, Gasserian and abdominal sympathetic ganglia. All the ganglia show histological lesions similar to those of the spinal cord and brain. Epidemic poliomyelitis is a general disease of the nervous system, although the most prominent and important symptoms are those following injury to the motor neurons of the spinal cord and brain. The virus of poliomyelitis is highly resistant to glycerin, in which it survives for more than two years; to 0.5 per cent. phenol, in which it survives for more than one year; while it succumbs after having been kept frozen constantly for several months.

The cerebrospinal fluid of convalescents tends to be devoid of the neutralizing immunity principles for the virus of poliomyelitis, although they may exceptionally be present within this fluid. Doubtless the immunity principles are not produced locally in the nervous tissues,

but elsewhere in the body, and are carried to the nervous organs by the blood.

Amoss (140) states that the globoid bodies or minute micro-organisms, cultivated from the central nervous organs of human beings and monkeys that have died of poliomyelitis, may be detected in the incubated brain tissues of infected monkeys in forms indicating post-mortem multiplication. Identical bodies have been detected in blood films prepared on the twelfth day of the acute attack from a paralyzed poliomyelitic monkey inoculated intraspinously.

The same organism has been cultivated from the blood of a monkey that has received intravenously a large dose of a Berkefeld filtrate of poliomyelitic virus. No other organism was detected, either in the sections of the brain or in film preparations of the blood. These observations tend, therefore, to confirm the etiological relationship between the minute micro-organisms and epidemic poliomyelitis suggested by the successful cultivation and inoculation experiments reported by Flexner and Noguchi. (124)

Clark and Amoss (141) state that by intraspinous injections of specimens of poliomyelitic virus of suitable virulence infection can be caused regularly in *Macacus rhesus* monkeys. The virus passes from the subarachnoid spaces into the nervous tissues in which it multiplies, and into the blood. The constant involvement of the pia-arachnoid membranes in poliomyelitis, even when no paralysis occurs, and the fact that infection can readily be produced by intraspinous inoculation, suggests anew that in the pathogenesis of poliomyelitis the interstitial tissue changes within the meninges, blood vessels and ground substance play a determining part.

While the virus injected into the subarachnoid spaces can be demonstrated there by inoculation tests forty-eight hours after the injection, it can no longer be detected on the sixth day, at a time when the first symptoms of infection make their appearance.

The failure of the cerebrospinal fluid from human and experimental cases of poliomyelitis to produce the disease when inoculated into monkeys is due to the fact that the virus is either fixed by the nervous tissue or passes into the blood.

BIBLIOGRAPHY.

Epidemiology.

1. The Recent Epidemic of Poliomyelitis, V. P. Gibney and Charlton Wallace. Jour. A. M. A., Vol. XLIX., No. 25, Dec. 21, 1907.

1*a*. The Early History of Infantile Poliomyelitis. Jour. A. M. A., Vol. LXII., No. 6, Feb. 7, 1914.

2. Epidemic Infantile Paralysis, M. Allen Starr. Jour. A. M. A., Vol. LI., No. 2, July 11, 1908.
3. Colmer. Am. Jour. Med. Sc. Han., 1843, p. 248.
3½. Leegaard. Norsk. Mag. F. Laegevidensk., Skaleen., 1907, LXVIII., 1241.
4. Bergenholtz. Reported by Medin, Verhandl. D. X. Internat. Med. Congress, Berlin, 1890.
5. Cordier. Lyon Med., Jan. 1, 1888.
6. Oxholm. Reported by Leegaard, Neurol. Centralbt., 1890, 760.
7. Medin. Nord. Med. Ark., 1896, No. 1.
8. Putnam, J. J. Boston Med. and Surg. Jour., Nov. 23, 1893.
9. André. Comp. rend. du Cong. de Méd. de Bordeaux., 1895, 352.
10. Caverly. New York Med. Record, Dec. 1, 1894; also MacPhail, Brit. Med. Jr., 1895.
11. Pieraccini. Lo. Sperimentale, Sept. 21, 1895.
12. Bucelli. Il. Policlinico, June 15, 1895.
13. Medin. Nord. Med. Ark., 1896, No. 1.
14. Altman. Australasian Med. Gaz., 1897. Lancet, July 3, 1897.
15. Pasteur. Trans. Clin. Soc., 1897, p. 143.
16. Taylor, J. M. Phila. Med. Jour., Jan. 29, 1898.
17. Bondurant. Phila. Med. News, Aug. 18, 1901.
18. Buzzard. Lancet, March 26, 1898.
19. Taylor, H. L. New York Med. Jour., 1897, p. 192.
20. Pleuss. Inaug. Diss., Kiel, 1898.
21. Newmark, L. Phila. Med. News, Jan. 28, 1899.
22. Packard, F. A. Jour. Nerv. and Ment. Dis., 1899, 210.
23. Auerbach. Jahrb. f. Kinderh., 1899; Neurol. Centralbt., 1900, 466.
24. Zappert. Jahrb. f. Kinderh., 1, III., p. 126.
25. Wickman. Heine-Medinschen Krankheit, Berlin, 1907, p. 148.
26. Leegaard. Norsk. Mag. f. Laegevidensk., 1901, 377.
27. Chapin, H. P. Archiv. Ped., 1900, 807.
28. McKenzie, D. H. New York Med. Rec., 1902, Vol. LXII., p. 528.
29. Painter, C. F. Tr. Am. Orthop. Assn., 1902, p. 414.
30. Woods, A. M. Occidental Med. Times, XVII., p. 77.
31. Wickman. Heine-Medinschen Krankheit, Berlin, 1907, p. 149.
32. Lorenzelli, A. La Pediatria, 1904, p. 428.
33. Blackhall, P. Australasian Med. Gaz., 1904, p. 347.
34. Ham, B. B. Australasian Med. Gaz., XXIV., p. 193.
35. Nannestad. Norsk. Mag. f. Laegevidensk., 1906, LXVII., 409.
36. Platon. Tidssk. f. d. Norsk. Laegefor., 1905, 601.
37. Giersvold. Norsk. Mag. f. Laegevidensk., 1905, 1280; Jahrb. f. Kinderh., 1906, LXIV., 255.
38. Wickman. Heine-Medinschen Krankheit, Berlin, 1907, p. 150.
39. Wickman. Heine-Medinschen Krankheit, Berlin, 1907, p. 262.
40. Lundgren, H. Hygeia, 1906, p. 1089.

41. Terriberry, Long Island Med. Jour., December, 1907.
42. Griffin. Jour. Mich. State Med. Soc., February, 1908, p. 142.
43. Free, S. M. Jour. Nerv. and Ment. Dis., April, 1908, p. 259.
44. Minnesota State Med. Assoc. Symposium, Jour. A. M. A., Vol. LIII., No. 21, Nov. 20, 1909.
45. An Analysis of 500 Cases of Spinal Infantile Paralysis, Jour. A. M. A., Vol. L., No. 22, May 30, 1908. George Collins and T. H. Romeiser.
46. Lovett, R. W. The Occurrence of Infantile Paralysis in Massachusetts in 1909, Monthly Bull. Mass. State Board of Health, June, 1910.
47. Epidemic in Westphalia. Jour. A. M. A., Vol. LIII., No. 19, Nov. 6, 1909, p. 1575.
48. The Epidemic of Spinal Disease in Nebraska. Jour. A. M. A., Vol. LIV., No. 4, Jan. 22, 1910. Shidler, G. P.
49. A Brief Report of the Nebraska Epidemic of Poliomyelitis, H. M. McClanahan. Jour. A. M. A., Vol. LV., No. 14, Oct. 1, 1910.
50. A Partial Report of the Epidemic of Acute Anterior Poliomyelitis in the State of Wisconsin during 1907–08, Jacolyn Manning. Wisconsin Med. Jour., April, 1909.
51. The Occurrence of Infantile Paralysis in Massachusetts in 1908, Lovett. Bost. Med. and Surg. Jour., July 22, 1909.
52. An Epidemic of Infantile Paralysis in Massachusetts in 1908. Emerson, H. C., Bost. Med. and Surg. Jour., July 22, 1909.
53. Epidemic Poliomyelitis, F. Kramer. Medizinische Klinik., Dec. 26, 1909.
54. Anterior Poliomyelitis. An Epidemic, Koplik. Archiv. of Ped., May, 1909, p. 321.
55. Acute Anterior Poliomyelitis. Le G. Kerr. Long Island Med. Jour., November, 1909.
56. Observations on the Occurrence of Epidemic Infantile Paralysis. Wollenweber Dortmund. Zeitzch. für Medizinalbeamt, No. 21, 1909.
57. The Increase of Acute Epidemic Infantile Paralysis in the Vicinity of Giessen. Berl. Klin. Wochensch., Dec. 27, 1909.
58. Acute Spinal Infantile Paralysis in the Rhenish-Westphalian Industrial District, Reckzeh. Medizin. Klinik, 1909. No. 45.
59. An Epidemic of Acute Poliomyelitis, W. W. Treves. Brain., 1909.
60. Spinal Paralysis in Children, F. Eichelberg. Deutsche Med. Wochenschrift, 1910, Vol. XXXVI., No. 3.
61. Appearance of Spinal Paralysis in Vorpommern, E. Peiper. Deutsche Med. Wochenschrift., 1910, Vol. XXXVI., No. 9.
62. The 1909 Epidemic of Acute Anterior Poliomyelitis (Epidemic Spinal Paralysis) in Nebraska, 1909. Jour. Am. Orthop. Assoc., Vol. VIII., No. 3, February, 1911.
63. Epidemic of Poliomyelitis in France and Elsewhere, A. Netter. Bull. of Academy of Med., Vol. LXIII., May 31, 1910, No. 21.
64. Studies in Infantile Paralysis during 1910, by the Washington State Board of Health. Pub. by the State.

65. Infantile Paralysis in Massachusetts during 1910. Reprint from the Monthly Bulletins of the Mass. State Board of Health for 1911.
66. The Epidemiology of Poliomyelitis, Joseph Collins. Jour. A. M. A., Vol. LIV., No. 24, June 11, 1910.
67. A Report of the Prevalence of Infantile Paralysis in Missouri, Archer O'Reilly. Jour. Missouri State Med. Assoc., November, 1911.
68. Acute Poliomyelitis in California, F. F. Gundrum. Jour. A. M. A., Jan. 27, 1912, Vol. LVIII., No. 4.
69. Sherman, H. M. Occid. Med. Times, August, 1897, p. 445.
70. Newmark, Leo. Med. News, 1899, LXXIV., 101.
71. Woods, A. M. Occid. Med. Times, March, 1903, p. 77.
72. Fleischner, E. C. Cal. State Jour. Med., September, 1911.
73. The Work of the Massachusetts State Board of Health in the Investigation of Infantile Paralysis, Robert W. Lovett. Bost. Med. and Surg. Jour., Vol. CLXVIII., No. 4, Jan. 23, 1913.
74. Domestic Animals and Epidemic Poliomyelitis, J. Bruno. Münch. Med. Wochenschrift., LX., No. 36, Sept. 9, 1913.
75. Epidemics of Poliomyelitis, Paul B. Roth. Lancet, Nov. 15, 1913.
76. Monthly Bulletin, Ohio State Board of Health. January, 1914, Vol. IV., No. 1, p. 23.
77. Epidemic Acute Poliomyelitis in Norway in the Years 1903 to 1906, Harbitz and Scheel. Jour. A. M. A., Vol. XLIX., No. 17, Oct. 26, 1907.
78. Acute Anterior Poliomyelitis, W. House. Northwest Med. Seattle, Washington, September, 1910.
79. Bulletin of the Kansas State Board of Health. July, 1910, Vol. VI., No. 7.
80. An Investigation as to the Occurrence in Massachusetts of Paralysis in the Lower Animals and Fowls, A. W. May. Monthly Bull., State Board of Health, Mass., June, 1911, Vol. 6, No. 6.
81. The Occurrence of Infantile Paralysis in the United States and Canada in 1910, Lovett. Am. Jour. Dis. of Children, August, 1911, Vol. 2.
82. Epidemic Poliomyelitis in Brazil, C. Ferreira. Bull. de la Soc. de Ped. Paris, October, XIII., No. 7.
83. Epidemiologic and Etiologic Studies of Acute Poliomyelitis in Pennsylvania, Dixon and Karsner. Am. Jour. Dis. of Children; October, 1911, Vol. 2.
84. Infantile Paralysis in 1910–11, editorial. Jour. A. M. A., Vol. LVIII., No. 18, p. 1379, May 4, 1912.
85. The Possible Etiological Relation of Certain Biting Insects to the Spread of Infantile Paralysis, Brues and Sheppard. Jour. of Economic Entomology, Vol. 5, No. 4, August, 1912.
173. Etiology of Epidemic Poliomyelitis, F. Lust and F. Rosenberg. Münchener Medizinische Wochenschrift., LXI., No. 3, Jan. 20, 1914.
174. Epidemic Poliomyelitis and Distemper of Dogs, R. H. Pierson. Jour. A. M. A., Vol. LXII., No. 9, Feb. 28, 1914.

Experimental.

86. The Microbe of Poliomyelitis, Harbitz and Scheel. Jour. A. M. A., Vol. L., No. 4, Jan. 25, 1908.
87. Editorial. Jour. A. M. A., June 19, 1909, Vol. LII., No. 25.
88. The Transmission of Acute Poliomyelitis to Monkeys, Flexner and Lewis. Jour. A. M. A., Vol. LIII., No. 20, 1909.
89. The Nature of the Virus of Epidemic Poliomyelitis, Flexner and Lewis. Jour. A. M. A., Vol. LIII., No. 25, p. 2095.
90. Experimental Epidemic Poliomyelitis in Monkeys, Flexner and Lewis. Jour. A. M. A., Vol. LIV., No. 14, April 2, 1910.
91. Experimental Poliomyelitis in Monkeys, Flexner and Lewis. Jour. A. M. A., May 18, 1910, p. 1780.
92. Experimental Investigations of Acute Epidemic Paralysis in Children, E. Meinicke. Deutsche Med. Wochenschrift., Vol. XXXVI., No. 15, 1909.
93. Etiology of Acute Poliomyelitis, H. Bonhoff. Deutsche Med. Wochenschrift., Vol. XXXVI., No. 12, 1910.
94. Etiology of Acute Epidemic Paralysis in Children, Kramer and Meinicke. Deutsche Med. Wochenschrift., Vol. XXXVI., No. 14, 1910.
95. Attempts to cultivate the Micro-organism of Infantile Paralysis, C. Levaditi. Presse Medicale, No. 6, 1910.
96. An Experimental Study of Acute Poliomyelitis, Landsteiner and Levaditi. Annales de L'Institut Pasteur, November, 1910, Vol. XXIV., No. 11.
97. Inoculations of Nasal Secretion from Patients with Acute Poliomyelitis. Jour. A. M. A., April 22, 1911.
98. Transmission of Acute Anterior Poliomyelitis to Monkeys, W. Knopfelmacher. Mediz. Klinik, No. 44, Oct. 31, 1909.
99. Experimental Research on Acute Anterior Poliomyelitis, C. Leiner and R. V. Weisner. Wiener Klin. Woch., Dec. 9, 1909, Vol. XXIII., No. 7.
100. Anterior Poliomyelitis. Etiology and Pathology. New Orleans Med. and Surg. Jour., March, 1910. Duval.
101. Experimental Studies on the Etiology of Acute Poliomyelitis, Strauss and Huntoon. New York Med. Jour., Jan. 8, 1910.
102. The Etiology of Acute Epidemic Infantile Paralysis, Krause and Meinicke. Deutsche Med. Wochenschrift., Oct. 21, 1909.
103. Acute Anterior Poliomyelitis with Autopsy, C. K. Russell. Montreal Med. Jour., December, 1909.
104. Epidemic Poliomyelitis in Monkeys, Flexner and Lewis. Jour. A. M. A., Vol. LIV., No. 1, Jan. 1, 1910.
105. The Diagnosis of Infantile Paralysis in the Prodromal and Early Acute Stage as found in the Experimental Study of Acute Poliomyelitis in Monkeys, Lucas. Trans. Mass. Med. Soc., June, 1910.

106. Anterior Poliomyelitis. Methods of Diagnosis from Spinal Fluid and Blood in Monkeys and in Human Beings, Gay and Lucas. Archiv. of Int. Med., Vol. 6, September, 1910.

107. Etiology of Poliomyelitis, F. Proescher. New York Med. Jour., Dec. 17, 1910.

108. Epidemic Poliomyelitis in Monkeys, Flexner and Lewis. Jour. A. M. A., Vol. LIV., No. 7, Feb. 12, 1910.

109. Acute Poliomyelitis, A. L. Skoog. Weekly Bull. Jackson County Missouri Med. Soc., Vol. 5, No. 1, Jan. 6, 1911.

110. Transmission Experiments with the Virus of Poliomyelitis, Osgood and Lucas. Jour. A. M. A., Vol. LVI., Feb. 18, 1911.

111. Abortive Cases of Poliomyelitis, Anderson and Frost. Jour. A. M. A., Vol. LVI., No. 9, March 4, 1911.

112. Experimental Poliomyelitis, Neustaedter and Thro. New York Med. Jour., Sept. 23, 1911.

113. Report of Committee on Methods for the Control of Epidemic Poliomyelitis. Jour. A. M. A., Vol. LVII., No. 16, Oct. 14, 1911.

114. Epidemic Poliomyelitis, Flexner and Clark. Jour. A. M. A., Nov. 18, 1911, Vol. LVII., No. 21.

115. Experiments as to the Protective Value of Certain Specific Sera and Vaccines against the Virus of Poliomyelitis, Lucas and Osgood. Report on Anterior Poliomyelitis, Mass. State Board of Health, 1911.

116. Transmission of Poliomyelitis, Pettersson. Jour. A. M. A., Vol. LIX., No. 14, Oct. 5, 1912.

117. Experimental Poliomyelitis in Monkeys. The Survival of the Poliomyelitis Virus in the Stomach and Intestine, Flexner, Clark and Dochez. Jour. A. M. A., Vol. LIX., No. 4, July 27, 1912.

118. Some Experimental Observations upon Monkeys concerning the Transmission of Poliomyelitis through the Agency of *Stomoxys calcitrans*, Rosenau and Brues. Monthly Bull., Mass. State Board of Health, September, 1912, Vol. 7, No. 9.

119. The Mode of Infection in Epidemic Poliomyelitis, Flexner. Jour. A. M. A., Vol. LIX., No. 15, Oct. 12, 1912.

120. Transmission of Poliomyelitis by Means of the Stable Fly (*Stomoxys calcitrans*). Public Health Reports, Oct. 25, 1912, p. 1733.

121. Possible Transmission of Poliomyelitis through the Dog, H. F. Langhorst. Jour. A. M. A., Vol. LIX., No. 26, Dec. 28, 1912.

122. Experiments on Insect Transmission of the Virus of Poliomyelitis, Howard and Clark. Jour. of Exp. Med., Vol. XVI., No. 6, December, 1912.

123. Epidemic Poliomyelitis. Passive Human Carriage of the Virus of Poliomyelitis, Flexner, Clark and Fraser. Jour. A. M. A., Vol. LX., No. 3, Jan. 18, 1913.

124. Experiments on the Cultivation of the Virus of Poliomyelitis, Flexner and Noguchi. Jour. A. M. A., Vol. LX., No. 5, Feb. 1, 1913.

125. Transmission to Monkeys of Virus obtained from English Cases of Poliomyelitis, McIntosh and Turnbull. Lancet, Feb. 22, 1913.
126. Transmission Experiments with the Virus of Poliomyelitis, Lucas and Osgood. Jour. A. M. A., Vol. LX., May 24, 1913.
127. Poliomyelitis and Variola, F. Proescher. New York Med. Jour., Vol. XCVII., No. 15, April 12, 1913.
128. The Mode of Transmission of Poliomyelitis, M. J. Rosenau. Trans. Mass. Med. Soc., 1913, p. 53.
129. Duration of Infectiousness of Secretions in Epidemic Poliomyelitis, Kling, Wernstedt and Pettersson. Zeitschrift für Immunitatsforschung., XVI., 1913, 17.
130. Attempts to transmit Poliomyelitis by Means of the Stable Fly (*Stomoxys calcitrans*), Sawyer, W. A., and Herms, W. B. Jour. A. M. A., LXI., No. 7, Aug. 16. 1913.
131. The Hypothesis of the Self-limitation of Poliomyelitis, Editorial. Jour. A. M. A., LXI., No. 21, Nov. 22, 1913.
132. Experimental Studies in Poliomyelitis, Dubois, Neal and Zingher. Jour. A. M. A., LXII., No. 1, Jan. 3, 1914.
133. Some Problems in Infection and its Control, S. Flexner. Science, Nov. 22, 1912. The Huxley Lecture.
134. Case of Acute Poliomyelitis associated with Diplococcal Infection of the Spinal Sac, Pasteur, Foulerton and MacCormac. Lancet, Feb. 15, 1908.
135. School as a Factor in Epidemic Poliomyelitis, J. Langer. Jahrbuch für Kinderheilkunde, LXXVI., No. 2, August, 1912.
136. Epidemic Poliomyelitis, Kling. Wien. Klin. Wochenschrift., XXVI., No. 2, Jan. 10, 1913.
137. Acute Anterior Poliomyelitis following Bite of Dog infected with Rabies, Haywood, J. B., Jr. New York Med. Jour., April 12, 1913.
138. A Contribution to the Epidemiology of Poliomyelitis, Flexner, Clark and Amoss. Jour. of Experimental Med., Vol. XIX., No. 2, Feb. 1, 1914.
139. A Contribution to the Pathology of Epidemic Poliomyelitis, Flexner, Clark and Amoss. Jour. of Exp. Med., Vol. XIX., No. 2, Feb. 1, 1914.
140. A Note on the Etiology of Epidemic Poliomyelitis, H. L. Amoss. Jour. Exp. Med., XIX., No. 2, Feb. 1, 1914.
141. Intraspinous Infection in Experimental Poliomyelitis, Clark and Amoss. Jour. Exp. Med., XIX., No. 2, Feb. 1, 1914.

II.

sgood, Dr. William P. Lucas, Dr. Arthur W. May, Dr. …

Charles T. Brues, instructor in economic entomology in Harvard University.

read at the Fifteenth International Congress on Hygiene and Demography, Washin
26, 1912. Reprinted from the Monthly Bulletin of the Massachusetts State Board of He
1912.

neighborhood of bodies of water may be shown by further investigation to be due to a greater prevalence in those districts of such in

Another fact brought out by the map of 1907 w

roads. There was, therefore, constant interchange of population these two districts, and yet in 1908 there were in the city of Springfield but 2 cases of infantile paralysis, and the intervening towns along the

Connecticut River showed at most 3 cases in Holyoke, 1 in Chicopee and 1 in Hatfield. In view of this experience it seems to me that whatever one may think of its contagiousness as affecting persons in immediate contact with patients, transfer of the infection by indirect contact through third persons must be very rare, if it ever occurs.

Another interesting point which has been noted by others is that a region once severely infected is not apt to be stricken during the succeeding year, and the Massachusetts maps of 1907 and 1908 show this phenomenon quite plainly. You will see that, for instance, in 1907 the Berkshire district had a considerable number of cases, whereas in 1908 there were very few. Furthermore, you will note that the Colrain district, which was severely affected in 1908, has been practically free from the disease ever since. In 1909 it is apparent that the Berkshire district again became severely affected, and again in 1910 the number of cases dropped off very markedly, even though the valley of the Connecticut River at this time showed a very large number of cases.

The experience in Massachusetts has been that the disease is less readily transmissible than scarlet fever, typhoid fever or diphtheria, but of course in such a comparison the abortive cases of infantile paralysis were not included. Even if such cases were included, however, I have little doubt that infantile paralysis, as compared with the diseases mentioned, is very much less contagious.

Another point which seems to stand out very sharply in the Massachusetts investigations is that the disease is very distinctly one of suburban or rural communities rather than one affecting more especially the cities. This statement rests upon the observations of 2,138 cases which have been analyzed in this regard for the years 1907–1910. The average population for the first 25 cities and towns most affected proved to be 5,205, whereas the average population of the 25 cities and towns least affected was 52,674, that is to say, cities and towns where the disease was relatively least frequent were ten times as large on the average as those where it was most frequent. As a control to this table cases of scarlet fever reported in the State for the year 1910 showed that in the 25 cities and towns in which scarlet fever was most prevalent the average population was 6,446, whereas in the 25 where it was least prevalent the average population was 7,633. In other words, there would seem to be some conditions radically different in the spread of infantile paralysis as compared with scarlet fever. This fact, taken in connection with the experience detailed above in the relation to cerebro-spinal meningitis, which disease, together with scarlet fever, is well known to be spread by contact with the nasopharyngeal secretions, must be given very weighty consideration when we come to estimate the rôle of these same

secretions in the spread of infantile paralysis; for the conditions favoring the transfer of these secretions, that is to say, the density of population, school attendance, overcrowding in wintertime and unhygienic surroundings — in other words, conditions found most prominently in city life — are not those which favor, apparently, the spread of infantile paralysis. Infantile paralysis, therefore, being in Massachusetts, at least, a country disease, one would look for some determining cause in country conditions as the reason for this apparent predilection for the rural districts, and as a result of investigation it is found that country children are exposed very much more strongly to any possible influence which animal disease might have upon them than city children. For instance, in the 25 cities and towns where the disease was least prevalent, that is to say, in the larger cities and towns, there was 1 cow to every 84 inhabitants and 1 horse to every 32 inhabitants; in the 25 cities and towns where the disease was most prevalent there was 1 cow to 11 inhabitants and 1 horse to 14 inhabitants. This table became very much more striking when a comparison was made of the numbers of swine, fowls and dogs. Now it is known that all these animals are subject at times to paralysis of varying types, and in a considerable number of instances paralysis in animals has been associated with paralysis in human beings. A considerable number, however, of paralyzed animals have been examined, and emulsions of their spinal cords have been injected into monkeys by Prof. Theobald Smith, but as yet with no positive results. In this connection, furthermore, it is apparent that country children are much more subject to the bites of insects than city children, and the possibility that insects may act as intermediate hosts for the virus of infantile paralysis, and may convey this virus from infected animals or infected human beings to other animals or human beings, must always be strongly borne in mind. In 1911 an investigation along this line of 88 cases in 17 cities and towns showed that in all instances the ordinary stable fly, *Stomoxys calcitrans,* was present in or about the house of the infected individual. Experiments looking to the possible infection of monkeys through the bites of this fly will be reported upon later by Professor Rosenau.

Meteorological records show that since 1904 Massachusetts has been subject to a constant deficiency rainfall. Such a deficiency would naturally be associated with a considerable increase in the amount of dust. When it is considered, however, that the disease has affected greatly other portions of the country in which there has been no such deficiency in rainfall the importance of this failure in the rain supply cannot be considered to be great.

An investigation as to the occurrence of this disease in institutions for

children showed that such children were much less liable to the disease than those leading an ordinary manner of life. They would seem to enjoy as a result of their somewhat complete isolation a freedom from infection.

Osgood and Lucas found an active virus in the nasopharyngeal membrane of the monkey five and a half months after an acute attack, and in the tonsils of a human being six months after an attack. These observations have naturally a very important bearing upon the question of contagion. In the first place, it suggests very strongly that the disease is transmitted by the secretions of the nasopharyngeal membranes, and, furthermore, that danger of contagion may persist for many months, and possibly longer; this in spite of the fact that Rosenau, Sheppard and Amoss failed to demonstrate the virus in the mouth and nose of 18 patients in various stages of the disease. It is, of course, well known that positive results have apparently been secured recently by Kling, Wernstedt and Pettersson. In other words, we have to do here, as in other infectious diseases, with the question of chronic carriers of disease and their relation to its propagation. Its importance becomes especially marked when we consider the number of cases which have been in contact with chronic cases of infantile paralysis previous to infection.

This persistence of the virus in the body of the infected individual may be important from another point of view, for there are a number of cases on record in the experience in Massachusetts in which the patient has apparently suffered from a second attack of the disease a few weeks, months or even years after the first attack. If such a second infection or reinfection may occur, it must be considered as possible that the patient between attacks may be a chronic carrier of the disease and therefore possibly responsible for secondary cases in others. Certain it is that very closely circumscribed localities may suffer from the disease over a considerable period of years. For instance, in one of the larger cities in Massachusetts, within a very small circumscribed area, 2 cases occurred in 1903, 2 cases in 1908, 1 case in 1909 and 1 case in 1910. The suggestion that a chronic carrier of infection was responsible for this situation is very strong. As is seen from the map for 1910 the city of Springfield, which up to that time had suffered a considerable and unexplained immunity from this disease, suffered from a quite severe epidemic. Furthermore, investigation as to the mortality from this and other acute diseases in said city showed very interesting results in that the mortality from cholera infantum, whooping cough and scarlet fever was also very much increased during this year. In fact, the mortality rate for cholera infantum, which for 1907, 1908 and 1909 had averaged 27, in 1910 jumped up to 106. The suggestion is, of

course, that a number of these cases of death reported as cholera infantum may have been and probably were typical cases of infantile paralysis of the gastro-intestinal type.

As far as therapeutics are concerned little new has been learned through the Massachusetts investigations. Osgood and Lucas, to be sure, made some experiments upon monkeys, with a certain number of specific sera and vaccines, to see whether the specific immunity brought about by these sera and vaccines might not give a partial immunity to infantile paralysis. The results, however, were negative. Hexamethylenamin has been recommended to the profession of Massachusetts strongly as a possible prophylactic against the disease, and quite generally employed.

A unique experiment with this drug was carried out at a certain boys' school in our State, an experiment which may be worthy of repetition by others under similar circumstances. At the opening of this school in the fall a boy arrived who had been in Europe and was in intimate contact with at least 25 of his fellow pupils for a period extending over ten days or two weeks. He then developed infantile paralysis, much to the discomfort of the school authorities. The boy was isolated immediately, and all the other pupils given hexamethylenamin in their drinking water. Whatever may have been the effect of this medication no other cases developed in this school. On the other hand, we have had one or two other similar experiences where cases in prodromal stages have been in intimate contact with school children and where no secondary cases have occurred, even though hexamethylenamin was not administered, — facts which make us very conservative in estimating the effect of simple contact in the spread of the disease.

As regards prognosis, the experience in Massachusetts has shown this to be much better than was previously supposed. In fact, the following conclusions seem justified: "In anterior poliomyelitis complete recovery or function recovery occurs in over 25 per cent. of cases examined at the end of four years. Atrophy may exist without impairment of function. In about one-half of the recovered cases the onset was mild. The distribution of the paralysis in such recovered cases was not essentially different from that in cases which do not recover. Recovery in many instances required months and in several cases from one to three years."

Another interesting possibility is that herpes zoster may be a form of anterior poliomyelitis, due to an unusual localization of the virus. Coincidence of this disease with epidemics of infantile paralysis has been noted, especially in recent years, by English observers. In the experience in Massachusetts certain striking cases have occurred. For

instance, in 1912 there has occurred, coincidently, in the same individua anterior poliomyelitis and herpes zoster. Furthermore, we have a hi tory, also, of anterior poliomyelitis in the child at the same time wi herpes zoster in the father. Pathologists maintain that the chang occurring in the posterior ganglia of the spinal cord in herpes zost resemble almost exactly those found in poliomyelitis in the anteri horns of the cord, and the hypothesis that the two diseases are due the same virus with different localizations is certainly one worthy further investigation.

Finally, the experience of Massachusetts has not been such as to su port the theory that infantile paralysis is spread from person to pers by direct or indirect contact. The rural preponderance of the diseas the comparative immunity of children confined in institutions and ho pitals, the summer incidence, the failure of the disease to find its greate incidence in cities and localities where density of population and ove crowding are most marked, and the irregular distribution have a militated against the acceptance of such a theory. In fact, the feelin among Massachusetts observers has been strong for some time that th epidemiology of this disease was best explained through the intermedia action of some biting insect, and evidence in support of this theory wi be presented by Prof. Milton J. Rosenau of Harvard University. (Se below.)

III.

SOME EXPERIMENTAL OBSERVATIONS UPON MONKEYS CONCERNING THE TRANSMISSION OF POLIOMYELITIS THROUGH THE AGENCY OF STOMOXYS CALCITRANS.[1]

A PRELIMINARY NOTE BY M. J. ROSENAU, M.D., PROFESSOR, PREVENTIVE MEDICINE AND HYGIENE, HARVARD MEDICAL SCHOOL, BOSTON, MASS., AND CHARLES T. BRUES, INSTRUCTOR IN ECONOMIC ENTOMOLOGY, BUSSEY INSTITUTION OF HARVARD UNIVERSITY.

The work we are about to report was done for, and under, the auspices of the State Board of Health of Massachusetts.

We should like to have it distinctly understood, and therefore, emphasize the fact right in the beginning, that this announcement is to be considered as a preliminary report, for the work is still in progress. Certain results have been obtained which it seems advisable to announce at this juncture. In taking this action in announcing work before it is completed we have not assumed the sole responsibility, but have taken counsel with older and wiser heads, friends for whose judgment we have the highest regard.

When we first took up the study of this disease — infantile paralysis — with the State Board of Health of Massachusetts, we considered all possible modes of transference of the virus from the sick to the well, but gradually focused our attention upon the fact that the disease seemed to be spread rather directly from person to person. In other words, the disease appeared to us at first blush to be a "contagious" disease, but one in which mild or abortive cases, missed cases, and third persons probably played an important rôle in the transfer of the infection. We were probably prejudiced in favor of this viewpoint on account of the splendid work of Wickman, whose publications we studied with care. We were further influenced to regard poliomyelitis as a "contagious" disease owing to the views of Flexner, who compared it to epidemic cerebro-spinal meningitis, and who regarded that it spread in the light of a contact infection through the secretions from the mouth and nose. The analogy to meningitis was a very close one, and the experimental fact that the virus could be demonstrated in the nasal mucosa of monkeys (Osgood, Lucas and others) seems to corroborate the suspicion that we are in fact dealing with an infection spread very much as cerebro-spinal meningitis is spread.

If these assumptions were correct then the virus should be demon-

[1] Remarks made by Professor Rosenau in the discussion of the previous paper.

strable in the secretions from the nose and throat. Rosenau, Sheppard and Amoss therefore injected 18 monkeys with the nasal and buccal secretions obtained from 18 persons who were suffering with the disease at the time, or in the stage of convalescence, or from persons suspected of acting as carriers. These results were negative. At the same time Straus of New York had a series of negative results, and other American workers were also unable to find the virus where we assumed it should be. These negative results seemed to us to have positive significance, and was the first definite indication that we were upon the wrong trail.

That poliomyelitis is not a "contagious" disease was clearly brought out by Dr. Richardson and other observers who have spoken this morning, all of whom have emphasized the point that the disease shows little or no tendency to spread in crowded districts, in schools, in institutions, in asylums, in camps and in other places where one would expect a disease spread by contact through secretions of the mouth and nose to spread most readily. We have in mind the fact that many cases of the disease have been brought into asylums and hospitals throughout the State of Massachusetts, in all stages of the infection; yet secondary cases have not occurred under such circumstances. On the contrary the disease prevailed in Massachusetts more particularly in rural and country districts sparsely settled.

Another reason that led us away from the theory of contacts, and made us believe that we were not dealing with a contagious disease in the ordinary sense of that term, was the close analogy between rabies and poliomyelitis. All investigators in laboratories who have worked with these two viruses have been struck with the similarity between rabies and poliomyelitis. Both viruses are diffused widely throughout the body, both exist in special concentration in the central nervous system, both are filterable, etc. Rabies being a wound infection made us conjecture that poliomyelitis may also be similarly transmitted.

Our experience with yellow fever, perhaps more than anything else, influenced us concerning the probable mode of transmission of poliomyelitis. It had been the privilege of one of us to work with yellow fever both before and after the mosquito days, and many analogies came to mind which made us believe that poliomyelitis also was not a contagious disease.

All the various reasons that influenced us in turning from contagion to some other mode of transference need not engage our attention now, for the history of this part of the work has been ably and accurately given by Dr. Richardson in the paper which he has just read. In justice to Dr. Richardson we desire to state that all the essential con-

clusions of his paper were arrived at before he knew of the results in the laboratory with the monkeys.

The work which we now briefly desire to report consists in exposing monkeys during all stages of the disease to the bites of *Stomoxys calcitrans.* The monkeys were infected in the usual way by bringing an emulsion of a known virus obtained from human sources in direct association with the central nervous system. After the flies had had abundant opportunity to bite these infected monkeys during the various stages of the disease, including the period of incubation, healthy monkeys were then exposed to the bites of these same flies. Of 12 healthy monkeys indications of the disease have been obtained in 6, 3 of them in a virulent form, resulting in death, the other 3 with transient tremblings, partial paralysis, diarrhœa and recovery. It is interesting to note that several of the monkeys had diarrhœa, therein the disease resembles the human disease more closely than when monkeys are simply inoculated with the virus into the brain, for gastro-intestinal upsets in children are frequently associated with infantile paralysis.

In these experiments it is important, we think, to use the proper technic in order to obtain successful results. The flies should be handled as little as possible. It is much better to handle the monkeys and leave the flies alone. In our experiment the flies were caught in nature, some of them were bred, placed in a large cage about 6 feet long by 5 or 6 feet wide, and some 3 or 4 feet high. The monkeys are stretched out at full length and wrapped in chicken wire. In this way they can be placed in the cage and the flies have full opportunity to bite. The flies appear to need a feed of blood about every day or two. They sometimes visit water which is kept in the cage, but apparently cannot be induced to eat any other food than the blood. At least, in our experiments, bananas, fruits and other substances exposed apparently were little visited by the flies. Furthermore, in our experiments a very large number of flies were used.

In conclusion we desire simply to summarize the fact that we have apparently transferred the virus of poliomyelitis from monkey to monkey through the bite of the stable fly, *Stomoxys calcitrans.* We would like to emphasize the fact that this does not appear to be simply a mechanical transference, but rather a biological one, requiring a period of extrinsic incubation in the intermediate host.

What conclusions can we draw from these facts? At present it seems to us we would not be justified in drawing any conclusion — the significance of the facts if confirmed is self-evident.

IV.

TRANSMISSION OF POLIOMYELITIS BY MEANS OF THE STABLE FLY (STOMOXYS CALCITRANS).[1]

By John F. Anderson, Director Hygienic Laboratory, and Wade H. Frost, Passed Assistant Surgeon, United States Public Health Service.

As a result of the thorough epidemiologic studies of poliomyelitis conducted by the Massachusetts State Board of Health from 1907 to 1912, under the direction of Dr. Mark W. Richardson, secretary of the board, evidence was collected which led the investigators to strongly suspect that the common stable fly (*Stomoxys calcitrans*) played an important part in the spread of this disease.

At the joint session of sections I. and V. of the Fifteenth International Congress on Hygiene and Demography in Washington, Sept. 26, 1912, Dr. Milton J. Rosenau, of the Harvard Medical School, who has been working in conjunction with the Massachusetts State Board of Health, announced the result of an experiment which seemed to confirm most strikingly the inferences drawn from the epidemiologic work above mentioned.

Dr. Rosenau stated that he had infected several monkeys with poliomyelitis by intracerebral inoculation, exposed them daily — from the time of inoculation till death — to the bites of several hundred *Stomoxys*, at the same time exposing 12 fresh monkeys to the bites of the same flies. At the time the announcement was made 6 of these 12 monkeys were reported as having developed symptoms characteristic of poliomyelitis, *i.e.*, illness followed by more or less extensive paralysis. Of these 6 monkeys 2 had died, 3 were paralyzed at that time, and 1 recovered after a brief illness. In the cord of one of the monkeys that had died were found the characteristic lesions of poliomyelitis, that is, perivascular infiltration and destruction of the motor cells of the anterior cornu. The cord of the other monkey was reported to have shown changes less characteristic of poliomyelitis, namely, degenerations of the motor cells without perivascular infiltration.

At the time of announcement a sufficient interval had not elapsed to determine the result of the attempt to transmit the infection to other monkeys by inoculation with the cord of one of the two that had died.

This experiment, giving an altogether new direction to the experi-

[1] Reprinted from Public Health Reports, Washington, D. C., Oct. 25, 1912.

mental study of poliomyelitis, appeared of sufficient importance to warrant an immediate attempt at confirmation.

In the experiment below reported it has been our object to repeat, as nearly as possible, the conditions of that reported by Dr. Rosenau, and we are indebted to him for assistance and advice in the details of the experiment.

On October 3, rhesus No. 242 was inoculated intracerebrally with an emulsion of the cord of a monkey which had died of poliomyelitis. The virus used is a strain originally obtained from the Rockefeller Institute for Medical Research, kept at the hygienic laboratory for nearly two years, during which time it has been passed through a large series of monkeys.

Two hours after inoculation the infected monkey was exposed to the bites of about 300 *Stomoxys* recently collected in Washington. Thereafter until death, on October 8, this animal was exposed daily for about two hours to the bites of the same flies, plus additional fresh *Stomoxys* added from time to time as caught. This monkey (No. 242) developed characteristic complete paralysis on the afternoon of October 7 and died at 2 A.M. October 8.

Another monkey (rhesus No. 246), similarly inoculated on October 5, was then exposed daily to the bites of the same flies, beginning October 7. This monkey developed paralysis on the morning of October 9, soon becoming completely paralyzed and dying that afternoon.

Thus, from October 4 to October 9, inclusive, the flies used had access to two monkeys inoculated with poliomyelitis, first, rhesus No. 242, then rhesus No. 246. It may be noted that the incubation period in both these monkeys was very short — four days from inoculation to the development of paralysis.

Beginning October 4, two fresh monkeys (rhesus No. 243 and Java No. 241) were exposed daily for about two hours to the bites of these same flies; and beginning October 5 a third fresh monkey (rhesus No. 244) was similarly exposed. All three of these animals subsequently developed symptoms of poliomyelitis, as follows: —

Java No. 241 was found completely paralyzed on the morning of October 12 and died a few hours later. At autopsy tubercles were found in the lungs, liver and spleen.

Rhesus No. 244 showed paralysis of the hind legs on the same day (October 12), but was, nevertheless, exposed again to the bites of the *Stomoxys* from 10 A.M. till 2 P.M. At 3 P.M. the animal, being almost completely paralyzed, was chloroformed. At autopsy tubercles were found in the lungs, liver and spleen, but apparently not sufficient to have been the cause of death.

[illegible]

[illegible]

[illegible]

[illegible] when a p[illegible] somewhat d[illegible] both fore legs were comp[illegible] the afternoon of the same [illegible] paralyzed, the left very weak, [illegible] died at 10.30 P.M. and was im[illegible] could be made at 9 A.M., Octobe[illegible] some congestion of the lower [illegible] marked on the left side, upon which the a[illegible] after paralysis developed. The meninges of the [illegible] were markedly congested. On section, the cord appeared edem[illegible] and the gray matter congested, showing minute hemorrhages. Th[illegible] of inoculation appeared normal except for a slight clot. Cultures [illegible] this site have shown no growth. The other organs were norm[illegible] appearance.

Histologic examination of the cord showed lesions characterist[illegible] poliomyelitis, intens[illegible] ion and perivascular infiltratio[illegible] round-cell infiltratio[illegible] l there in the gray matt[illegible] the cells of the ant[illegible], and small hemo[illegible] and posterior cornu.

Conclusion.

These results, in confirmation of those announced by Dr. Rosenau, would seem to demonstrate conclusively that poliomyelitis may be transmitted to monkeys through the agency of the stable fly (*Stomoxys calcitrans*).

It remains for further work to decide whether this is the usual or the only method of transmission in nature.

V.

FURTHER EXPERIMENTS IN POLIOMYELITIS.

By M. J. Rosenau, M.D., Department of Preventive Medicine and Hygiene, Harvard University Medical School, Boston.

Experiments with the Stable Fly. — During the fall of 1912 attempts were made to obtain further information concerning the transmission of the virus of poliomyelitis through the stable fly. A large number of experiments were conducted with a modified technique. The flies, a few in number, were kept in battery jars and applied to the monkeys from time to time. Frequently, only one fly was made the subject of experiment, with the hope that something might be learned concerning the period of incubation and other factors. All these experiments, however, resulted negatively.

Another series of experiments were started in July, 1913, and consisted in exposing infected monkeys on alternate days to the bites of numerous stable flies kept in large cages and under conditions similar to those of the summer of 1912. Four separate cages were used. Flies caught in nature were placed in three of the cages. The remaining cage, No. 4, was used to hold flies bred by Professor Brues at the Bussey Institution. These experiments also resulted negatively.

The details of the experiments are plainly seen in the tables: —

Fly Cage No. 1.

[The number of flies is approximate. + = monkey exposed to fly bites; I = incubation period; S = symptoms; P = paralysis.]

	1913.																
	July 30.	July 31.	Aug. 1.	Aug. 2.	Aug. 3.	Aug. 4.	Aug. 5.	Aug. 6.	Aug. 7.	Aug. 8.	Aug. 9.	Aug. 10.	Aug. 11.	Aug. 12.	Aug. 13.	Aug. 14.	Aug. 15.
Infected rhesus No. 85, ...	+ I	+ I	+ I	+ I	+ P	+ P	+ P	+ P	+ P	+ P		+ P				+ P	
Infected rhesus No. 162, ...								+ I	+ I		+ S	+ P					
Infected rhesus No. 47, ...														+ I		+ I	
Infected rhesus No. 83, ...																	
Infected rhesus No. 19, ...																	
Infected rhesus No. 168, ...																	

Fly Cage No. 1 — Continued.

	1913.																
	July 30.	July 31.	Aug. 1.	Aug. 2.	Aug. 3.	Aug. 4.	Aug. 5.	Aug. 6.	Aug. 7.	Aug. 8.	Aug. 9.	Aug. 10.	Aug. 11.	Aug. 12.	Aug. 13.	Aug. 14.	Aug. 15.
Normal rhesus No. 86, ...						+	+	+	+	+	+		+		+		+
Normal rhesus No. 87, ...							+	+	+	+	+		+		+		+
Normal rhesus No. 88, ...										+	+		+		+		+
Number of flies added to cage, ...	200	25	200	410	200	300	400	400	800	800	800	-	-	-	-	-	-
Number of flies that fed, ...	25	75	150	300	400	700	700	600	800	800	600	800	900	800	500	500	few

Fly Cage No. 1 — Concluded.

	1913.															
	Aug 16.	Aug. 17.	Aug. 18.	Aug. 19.	Aug. 20.	Aug. 21.	Aug. 22.	Aug. 23.	Aug. 24.	Aug. 25.	Aug. 26.	Aug. 27.	Aug. 28.	Aug. 29.	Aug. 30.	Aug. 31.
Infected rhesus No. 85,																
Infected rhesus No. 162,																
Infected rhesus No. 47,	+ P		+ P													
Infected rhesus No. 83,	+ P															
Infected rhesus No. 19					+ I		+ P									
Infected rhesus No. 168,									+ P							
Normal rhesus No. 86,		+		+		+					+			+		
Normal rhesus No. 87,		+		+				+				+			+	
Normal rhesus No. 88,		+		+						+			+			+
Number of flies added to cage,	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Number of flies that fed,	few	few	150	60	60	60	40	40	30	10	6	4	2	5	4	4

Fly Cage No. 2.

[The number of flies is approximate. +=monkey exposed to fly bites; I=incubation period; S=symptoms; P=paralysis.]

	1913.																		
	Aug. 11.	Aug. 12.	Aug. 13.	Aug. 14.	Aug. 15.	Aug. 16.	Aug. 17.	Aug. 18.	Aug. 19.	Aug. 20.	Aug. 21.	Aug. 22.	Aug. 23.	Aug. 24.	Aug. 25.	Aug. 26.	Aug. 27.	Aug. 28.	[illegible]
Infected rhesus No. 85,	+ P	+ P			+ P														
Infected rhesus No. 162,	+ P																		
Infected rhesus No. 47,					+ S				+ P										
Infected rhesus No. 83,							+ P												
Infected rhesus No. 19,											+ P								
Infected rhesus No. 36,											+ S								
Infected rhesus No. 76,													+ I				+ P		
Infected rhesus No. 65,													+ I		+ P		+ P		

Infected rhesus No. 168,															+ P		+ P		
Infected rhesus No. 67,																			
Infected rhesus No. 32,																			
Infected rhesus No. 84,																			
Infected rhesus No. 107,																			
Normal rhesus No. 90,			+	+		+		+	Diarrhœa; died August 29; autopsy showed death due to infection of intestinal tract.										
Normal rhesus No. 91,			+	+		+		+		+		+		+		+		+	
Normal rhesus No. 97,										+		+		+		+		+	
Number of flies added to cage,	800	300	1,000	630	200	300	-	-	200	-	-	1,000	-	-	600	1,500	-	-	-
Number of flies that fed,	100	500	800	900	1,000	800	-	500	500	400	-	1,200	800	900	800	900	800	600	800

Fly Cage No. 2 — Continued.

	1913.																	
	Aug. 30.	Aug. 31.	Sept. 1.	Sept. 2.	Sept. 3.	Sept. 4.	Sept. 5.	Sept. 6.	Sept. 7.	Sept. 8.	Sept. 9.	Sept. 10.	Sept. 11.	Sept. 12.	Sept. 13.	Sept. 14.	Sept. 15.	Sept. 16.
Infected rhesus No. 85, ...																		
Infected rhesus No. 162, ...																		
Infected rhesus No. 47, ...																		
Infected rhesus No. 83, ...																		
Infected rhesus No. 19, ...																		
Infected rhesus No. 36, ...																		
Infected rhesus No. 76, ...																		
Infected rhesus No. 65, ...																		

Infected rhesus No. 168, ...																		
Infected rhesus No. 67, ...		+S		+P		+P												
Infected rhesus No. 32, ...				+S		+P												
Infected rhesus No. 84, ...								+S		+P								
Infected rhesus No. 107, ...																		
Normal rhesus No. 90, ...																		
Normal rhesus No. 91, ...	+		+		+		+		+		+		+		+		+	
Normal rhesus No. 97, ...	+		+		+		+		+			+		+		+		+
Number of flies added to cage, ...	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Number of flies that fed, ...	-	-	-	-	500	400	300	200	-	100	75	50	-	40	30	20	10	10

Fly Cage No. 2 — Concluded.

	1913.																	
	Sept. 17.	Sept. 18.	Sept. 19.	Sept. 20.	Sept. 21.	Sept. 22.	Sept. 23.	Sept. 24.	Sept. 25.	Sept. 26.	Sept. 27.	Sept. 28.	Sept. 29.	Sept. 30.	Oct. 1.	Oct. 2.	Oct. 3.	Oct. 4.
Infected rhesus No. 85, ...																		
Infected rhesus No. 162, ...																		
Infected rhesus No. 47, ...																		
Infected rhesus No. 83, ...																		
Infected rhesus No. 19, ...																		
Infected rhesus No. 36, ...																		
Infected rhesus No. 76, ...																		
Infected rhesus No. 65, ...																		

Infected rhesus No. 168, ...																		
Infected rhesus No. 67, ...																		
Infected rhesus No. 32, ...																		
Infected rhesus No. 84, ...																		
Infected rhesus No. 107, ...												† I		† P				
Normal rhesus No. 90, ...																		
Normal rhesus No. 91, ...	+		+		+		+		+		+		+		+		+	
Normal rhesus No. 97, ...		+		+		+		+		+						+		+
Number of flies added to cage, ...	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Number of flies that fed, ...	10	10	8	8	6	6	8	8	6	6	6	6	6	4	5	5	4	4

Fly Cage No. 3 — Flies Bred.

[The number of flies is approximate. + = monkey exposed to fly bites; I = incubation period; S = symptoms; P = paralysis.]

	1913.																		
	Aug. 15.	Aug. 16.	Aug. 17.	Aug. 18.	Aug. 19.	Aug. 20.	Aug. 21.	Aug. 22.	Aug. 23.	Aug. 24.	Aug. 25.	Aug. 26.	Aug. 27.	Aug. 28.	Aug. 29.	Aug. 30.	Aug. 31.	Sept. 1.	Sept. 2.
Infected rhesus No. 83, ...	+S																		
Infected rhesus No. 47, ...			+P			+P													
Infected rhesus No. 73, ...			+P	+P															
Infected rhesus No. 36, ...						+I		+S											
Infected rhesus No. 65, ...								+I		+I		+P							
Infected rhesus No. 67, ...																+S		+P	
Infected rhesus No. 76, ...								+I		+I		+P		+P		+P			
Infected rhesus No. 32, ...																		+I	

Infected rhesus No. 56,																			
Infected rhesus No. 168,						+I		+I				+P							
Infected rhesus No. 64,																			
Infected rhesus No. 63,																			
Infected rhesus No. 106,																			
Infected rhesus No. 107,																			
Normal rhesus No. 92,		+			+	+	+	Died; tuberculosis.											
Normal rhesus No. 94,					+	Diarrhœa.					+		+		+	Diarrhœa.			
Normal rhesus No. 95,					+		+		+	+	+		+	+	+	+	+		+
Normal rhesus No. 96,					+		+		+	+	+	+	+	+	+		+	+	+
Normal rhesus No. 98,									+		+		+		+	+	+		+
Number of flies added to cage,	600	600	-	1,000	400	400	-	-	-	-	-	-	-	-	-	-	-	-	-
Number of flies that fed,	500	900	-	1,200	1,500	2,000	1,800	-	1,800	1,800	1,600	1,600	1,500	1,000	1,000	1,000	-	800	800

Fly Cage No. 3 — Flies Bred — Continued.

	1913.																		
	Sept. 3.	Sept. 4.	Sept. 5.	Sept. 6.	Sept. 7.	Sept. 8.	Sept. 9.	Sept. 10.	Sept. 11.	Sept. 12.	Sept. 13.	Sept. 14.	Sept. 15.	Sept. 16.	Sept. 17.	Sept. 18.	Sept. 19.	Sept. 20.	Sept. 21.
Infected rhesus No. 83, ...																			
Infected rhesus No. 47, ...																			
Infected rhesus No. 73, ...																			
Infected rhesus No. 36, ...																			
Infected rhesus No. 65, ...																			
Infected rhesus No. 67, ...	+ P		+ P																
Infected rhesus No. 76, ...																			
Infected rhesus No. 32, ...	+ P		+ P																

Infected rhesus No. 56,	+ P																		
Infected rhesus No. 168,																			
Infected rhesus No. 64,					+ I														
Infected rhesus No. 63,					+ I		+ S												
Infected rhesus No. 106,																			
Infected rhesus No. 107,																			
Normal rhesus No. 92,																			
Normal rhesus No. 94,																			
Normal rhesus No. 95,																			
Normal rhesus No. 96,		+		+		+		+		+		+		+		+		+	
Normal rhesus No. 98,		+		+		+			+		+		+		+		+		+
Number of flies added to cage,	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Number of flies that fed,	800	400	300	400	-	300	250	150	75	60	70	40	20	20	20	20	10	10	5

Fly Cage No. 3 — Flies Bred — Concluded.

	1913.																		
	Sept. 22.	Sept. 23.	Sept. 24.	Sept. 25.	Sept. 26.	Sept. 27.	Sept. 28.	Sept. 29.	Sept. 30.	Oct. 1.	Oct. 2.	Oct. 3.	Oct. 4.	Oct. 5.	Oct. 6.	Oct. 7.	Oct. 8.	Oct. 9.	Oct. 10.
Infected rhesus No. 83,																			
Infected rhesus No. 47,																			
Infected rhesus No. 73,																			
Infected rhesus No. 36,																			
Infected rhesus No. 65,																			
Infected rhesus No. 67,																			
Infected rhesus No. 76,																			
Infected rhesus No. 32,																			

Infected rhesus No. 56,																			
Infected rhesus No. 168,																			
Infected rhesus No. 64,																			
Infected rhesus No. 63,																			
Infected rhesus No. 106,					+ I														
Infected rhesus No. 107,									+ P										
Normal rhesus No. 92,																			
Normal rhesus No. 94,																			
Normal rhesus No. 95,																			
Normal rhesus No. 96,	+		+			+		+		+		+			+		+		+
Normal rhesus No. 98,		+		+			+				+		+			+		+	
Number of flies added to cage,	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Number of flies that fed,	5	8	8	8	8	8	8	6	2	5	5	5	4	-	2	2	2	2	1

Fly Cage No. 4 — Flies caught in Nature.

[The number of flies is approximate. + = monkey exposed to fly bites; I = incubation period; S = symptoms; P = paralysis.]

	1913.																					
	Aug. 27.	Aug. 28.	Aug. 29.	Aug. 30.	Aug. 31.	Sept. 1.	Sept. 2.	Sept. 3.	Sept. 4.	Sept. 5.	Sept. 6.	Sept. 7.	Sept. 8.	Sept. 9.	Sept. 10.	Sept. 11.	Sept. 12.	Sept. 13.	Sept. 14.	Sept. 15.	Sept. 16.	Sept. 17.
Infected rhesus No. 67, .	+ I	+ I																				
Infected rhesus No. 99, .		+ I		+ P																		
Infected rhesus No. 168, .		+ P		+ P																		
Infected rhesus No. 56, .						+ I																
Infected rhesus No. 84, .								+ I		+ I		+ P										
Infected rhesus No. 164, .								+ I		+ I		+ I		+ I		+		+		+		+
Infected rhesus No. 63, .																+ I						
Infected rhesus No. 40, .																+ S						
Infected rhesus No. 106, .																						

Infected rhesus No. 107, .																						
Infected rhesus No. 112, .																						
Infected rhesus No. 68, .																+ I		+ S				
Infected rhesus No. 105, .																						
Normal rhesus No. 100, .			+		+		+		+		+		+		+		+		+		+	
Normal rhesus No. 101, .			+	+	+		+		+		+		+		+		+		+		+	
Normal rhesus No. 102, .			+		+	+	+		+		+		+				+		+		+	
Normal rhesus No. 103, .			+	+	+		+		+		+		+				+		+		+	
Normal rhesus No. 108, .																						
Normal rhesus No. 109, .																						
Normal rhesus No. 110, .																						
Normal rhesus No. 111, .																						
Number of flies added to cage.	1,700	600	-	-	-	-	800	-	-	-	-	-	-	400	800	-	-	-	-	-	-	200
Number of flies that fed, .	200	500	800	800	-	500	900	900	700	600	700	-	700	800	900	-	1,000	900	800	200	200	150

Fly Cage No. 4 — Flies caught in Nature — Continued.

	1913.																					
	Sept. 18.	Sept. 19.	Sept. 20.	Sept. 21.	Sept. 22.	Sept. 23.	Sept. 24.	Sept. 25.	Sept. 26.	Sept. 27.	Sept. 28.	Sept. 29.	Sept. 30.	Oct. 1.	Oct. 2.	Oct. 3.	Oct. 4.	Oct. 5.	Oct. 6.	Oct. 7.	Oct. 8.	Oct. 9.
Infected rhesus No. 67, .																						
Infected rhesus No. 99, .																						
Infected rhesus No. 168, .																						
Infected rhesus No. 56, .																						
Infected rhesus No. 84, .																						
Infected rhesus No. 164, .		+		+		+		+		+												
Infected rhesus No. 63, .																						
Infected rhesus No. 40, .																						
Infected rhesus No. 106, .								+ I		+ S												

Infected rhesus No. 107, .										+ I		+ S										
Infected rhesus No. 112, .													+ S									
Infected rhesus No. 68, .																						
Infected rhesus No. 105, .		+ S		+ P		+ P																
Normal rhesus No. 100, .	+		+		+		+															
Normal rhesus No. 101, .	+		+		+		+															
Normal rhesus No. 102, .	+		+		+		+															
Normal rhesus No. 103, .	+		+		+		+															
Normal rhesus No. 108, .									+		+		+		+	+		+		+		+
Normal rhesus No. 109, .									+		+		+		+		+		+		+	
Normal rhesus No. 110, .									+		+		+		+		+		+		+	
Normal rhesus No. 111, .									+		+		+		+			+		+		+
Number of flies added to cage.	1,000	-	-	-	-	600	600	600	-	1,000	-	-	-	-	-	-	-	-	-	-	-	-
Number of flies that fed, .	200	250	600	-	200	200	800	1,200	800	800	800	400	400	400	300	300	300	400	450	400	400	300

Fly Cage No. 4 — Flies caught in Nature — Concluded.

	1913.																					
	Oct. 10.	Oct. 11.	Oct. 12.	Oct. 13.	Oct. 14.	Oct. 15.	Oct. 16.	Oct. 17.	Oct. 18.	Oct. 19.	Oct. 20.	Oct. 21.	Oct. 22.	Oct. 23.	Oct. 24.	Oct. 25.	Oct. 26.	Oct. 27.	Oct. 28.	Oct. 29.	Oct. 30.	Oct. 31.
Infected rhesus No. 67, .																						
Infected rhesus No. 99, .																						
Infected rhesus No. 168, .																						
Infected rhesus No. 56, .																						
Infected rhesus No. 84, .																						
Infected rhesus No. 164, .																						
Infected rhesus No. 63, .																						
Infected rhesus No. 40, .																						
Infected rhesus No. 106, .																						

Infected rhesus No. 107, .																						
Infected rhesus No. 112, .																						
Infected rhesus No. 68, .																						
Infected rhesus No. 105, .																						
Normal rhesus No. 100, .																						
Normal rhesus No. 101, .																						
Normal rhesus No. 102, .																						
Normal rhesus No. 103, .																						
Normal rhesus No. 108, .		+		+		+		+		+				+				+				
Normal rhesus No. 109, .	+		+		+		+		+		+				+				+			
Normal rhesus No. 110, .	+		+		+		+		+			+				+				+		
Normal rhesus No. 111, .		+		+		+		+					+				+				+	
Number of flies added to cage.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Number of flies that fed, .	300	200	200	100	100	75	50	50	20	20	20	15	15	10	10	10	10	10	8	5	1	

Feeding Experiments. — A number of experiments were made in order to infect monkeys by the mouth. The animals were fed with the virus in various foods and drink, such as banana and milk. The virus was usually given on an empty stomach, and was given to both normal monkeys and monkeys with diarrhœa. As soon as one of the stock monkeys showed symptoms of diarrhœa he was at once isolated and fed with the virus, with the expectation that perhaps the digestive disturbance would prompt the virus to penetrate the intestinal mucosa. All the feeding experiments resulted negatively.

Infection through the Nasal Mucosa. — Several attempts were made to infect monkeys through the uninjured nasal mucosa, with only one positive result, as follows: —

Rhesus No. 91. Previously exposed to Flies, with Negative Results.

Dec. 2. About 1 cubic centimeter of a very rich emulsion of the virus (brain and spinal cord of rhesus No. 107) was placed in the nostrils while the animal was under ether. The monkey did not cough or sputter, and probably swallowed most of the virus.

Dec. 10. Trembling; poor appetite.

Dec. 11. Trembling more evident, especially on right side; poor appetite; no paralysis.

Dec. 12. Paresis, right side, which soon became more marked with definite paralysis of right arm and right leg; recovered with residual paralysis.

Quinine in Poliomyelitis. — In view of the favorable results reported by the use of quinine in rabies, a few attempts were made to determine the influence of this drug upon poliomyelitis. Although nothing encouraging developed from the few experiments in which quinine bihydrochlorid was used, both as a prophylactic and therapeutic agent, nevertheless, it must be remembered that the monkeys were given an overpowering amount of the virus directly into the brain tissue. It is possible that with less overwhelming amounts of infection, and with more normal channels of entrance, different results may be obtained.

It is interesting to note that a normal monkey was able to withstand 0.1 of a gram of quinine bihydrochlorid intravenously, whereas monkeys just beginning to show symptoms of poliomyelitis died at once with this same amount. The following protocols are given: —

Rhesus No. 102. Previously exposed to Flies, with Negative Results.

Nov. 10. At 11.55 A.M. 0.2 cubic centimeter emulsion of virus No. 107 intracranially; 0.1 gram quinine bihydrochlorid intravenously at same time. At 4.25 P.M. 0.1 gram of quinine bihydrochlorid subcutaneously.

Nov. 11. At 9.15 A.M. 0.1 gram of quinine bihydrochlorid subcutaneously. At 12.45 P.M. 0.1 gram of quinine bihydrochlorid subcutaneously. At 4.45 P.M. 0.1 gram of quinine bihydrochlorid subcutaneously.

Nov. 12. At 9.05 A.M. 0.1 gram of quinine bihydrochlorid subcutaneously. At 12.45 P.M. 0.1 gram of quinine bihydrochlorid subcutaneously. At 4.45 P.M. 0.1 gram of quinine bihydrochlorid subcutaneously.

Nov. 13. At 9 A.M. 0.1 gram of quinine bihydrochlorid subcutaneously. At 12.30 P.M. 0.1 gram of quinine bihydrochlorid subcutaneously. At 5.45 P.M. 0.2 gram of quinine bihydrochlorid subcutaneously.

Nov. 14. Well marked tremor; weakness of left wrist and legs.

Nov. 15. Complete paralysis; died at 8.45 P.M.

Rhesus No. 88. Previously exposed to Flies, with Negative Results.

Nov. 10. 0.2 cubic centimeter emulsion of virus No. 107 intracranially.

Nov. 14. Somewhat more quiet than usual.

Nov. 15. Very quiet; very slight tremor; some difficulty in walking. At 9.45 A.M. 0.3 gram quinine bihydrochlorid intravenously; died at once.

Rhesus No. 96. Previously exposed to Flies, with Negative Results.

Nov. 10. 0.2 cubic centimeter emulsion of virus No. 107 intracranially.

Nov. 14. Unusually quiet; poor appetite.

Nov. 15. Marked trembling; 0.1 gram of quinine bihydrochlorid intravenously; died at once.

Rhesus No. 95. Previously exposed to Flies, with Negative Results.

Nov. 10. 0.2 cubic centimeter emulsion of virus No. 107 intracranially.

Nov. 12. Marked trembling; rather quiet; 0.1 gram quinine bihydrochlorid intravenously; died at once.

VI.

THE MODE OF TRANSMISSION OF POLIOMYELITIS.[1]

By M. J. Rosenau, M.D., Department of Preventive Medicine and Hygiene, Harvard University Medical School, Boston.

From a practical point of view the mode of transmission is the most useful single factor in combating a disease. The health officer would prefer to know the precise mode or modes of transmission of any disease rather than its cause or pathologic anatomy or even its treatment. Hence a large amount of work has been done to determine how the virus of poliomyelitis leaves the body, how it enters its victim, and the route it takes from one person to the next. Despite all the thought and work that has been focused on this problem, the mode of transmission of poliomyelitis remains an open chapter, and although much light has been thrown on the subject, the present state of our knowledge does not permit of dogmatic, much less final, statements.

There are two avenues of approach to a problem of this sort; one, through epidemiologic field studies, and the other through laboratory research work. Both these trails have been blazed. Much of the epidemiologic work has given conflicting results, and much of the laboratory work has likewise been confusing. The evidence obtained from the field and that from the laboratory, however, do not have an equal standing before the court. The fallibility of epidemiologic evidence has long been recognized. Sanitarians who have had a long experience know full well that it has always been necessary to revise the chapter on the epidemiology of a disease as soon as its mode of transference is discovered. All those who have collected field data are fully aware of the pitfalls. Errors are unavoidable from the very nature of the circumstances; the personal equation and also the limitations of the investigator often warp or dwarf the important facts. In any event, the enormous mass of data collected by careful investigators in the field is perplexing and difficult to analyze. Even if the epidemiologist has the detective instinct of a Sherlock Holmes, and the statistical genius of a Karl Pearson, he may be wholly carried off the track by the missed cases, and by the carriers, or by an incomplete knowledge of unusual forms of the disease, or by unknown factors in its etiology. On the other hand, the exact observations from the laboratory often throw a flood of light on our field work, and when the two are correlated we have real and useful additions to our knowledge.

[1] Reprinted from the Journal of the American Medical Association, May 24, 1913, Vol. LX, pp. 1612-1615.

There are many theories to account for the spread of poliomyelitis. The chief ones may be summarized under four headings: (1) that it is a "contagious" disease, communicated directly from person to person through the secretions from the mouth and nose; (2) that it is an insect-borne disease; (3) that it is conveyed through dust; (4) that it is an alimentary infection, the virus being taken in with food and drink and absorbed from the digestive tube. There is evidence from the field and from the laboratory to support each one of these theories. These four theories, however, do not include all the views brought forward to explain the mode of transmission of infantile paralysis. For example, the hypothesis has been expressed that the disease is transmitted to man from lower animals, particularly domesticated animals, but there has been no convincing demonstration that the infection occurs naturally in any other animal than man.

In view of the uncertainty concerning the mode of transmission of poliomyelitis it is worth while to take stock of our knowledge of this subject. I have therefore briefly summarized the evidence pro and con.

That anterior poliomyelitis may be a contagious disease was first announced by Wickman of Sweden, whose epidemiologic investigations upon the subject are now classic. Wickman formulated a new symptomatology; his greatest contribution to the subject was, perhaps, the discovery that abortive cases of the disease occur. These mild and hitherto unrecognized clinical forms shed an entirely different light on the epidemiology. Wickman brought forward strong evidence in support of the view that the disease was transmitted directly from person to person, especially through the abortive or missed cases, as well as through suspected carriers.

The theory that infantile paralysis is a "contagious" disease was the first, and is the most natural, explanation to account for its spread. In many respects infantile paralysis resembles epidemic cerebrospinal meningitis, with which disease it is, in fact, sometimes confused. Flexner has emphasized the resemblance between the two infections, and has been an able and valiant champion of the view that the virus in both these diseases leaves the body in the secretions from the mouth and nose and enters the victim through the same channel. Corroboration of this view has come especially through the work of the Swedish investigators, Kling, Pettersson and Wernstedt, who claim to have demonstrated the virus in the secretions from the nose and throat, not only in cases during the acute stage, but also during various stages of convalescence, and even in healthy carriers. Furthermore, Osgood and Lucas demonstrated the virus in the mucous membrane of a monkey five and a half months after recovery from the experimental disease, and recently they recovered it from a

chronic carrier in man. Kling claims to have found the virus in the nasopharynx up to seven months in a few human cases. Finally, it is possible to infect monkeys simply by placing the virus on the uninjured mucous membrane of the nose and throat. The evidence, therefore, that we are dealing with an infection that leaves the body in the secretions from the nose and throat, and enters by the same channel, is strong, both from epidemiologic studies and from laboratory investigations. That this evidence, however, is not conclusive, may be gleaned by a little closer consideration of the facts.

It is important, first of all, to remember that we have no clear-cut criterion by which to judge what is and what is not anterior poliomyelitis. When the symptoms are characteristic and the lesions typical we are justified in making a definite diagnosis. In any critical case, however, further corroboration must be had by transferring the virus from monkey to monkey in order to demonstrate that we are, in fact, dealing with a communicable infection capable of reproducing itself. When we apply this criterion to some of the work that has been reported, to support the view that infantile paralysis is a "contagious" disease, we find the evidence not as strong as the conclusions of the experimenters would lead us to suspect. The mere fact that an animal has an acute paralytic affection, associated with perivascular infiltrations, degeneration of the neurons, and occasional hemorrhages into the cord, is not enough, in the present state of our knowledge, to justify us in labeling it poliomyelitis. The criterion in critical cases must be the power of the virus to reproduce itself and repeat the symptoms and lesions of the infection through succeeding generations.

There has been a long series of negative results in attempts to demonstrate the presence of the virus in the secretions from the mouth and nose. Strauss, Rosenau, Shepard and Amoss, Flexner, and also Anderson and others, have all reported failures in this regard. These negative results have a certain degree of positive significance, for if the usual means of transmission of the disease is through the secretions from the mouth and nose we should not have such great difficulty in demonstrating its presence in these secretions. Only Kling,[1] Pettersson and Wernstedt have found it comparatively easy to make this demonstration. These investigators believe that they have established the important fact that

[1] Kling found it comparatively easy to demonstrate the presence of the virus in the washings from the mucous membranes. Thus he found that 78 per cent. of the monkeys contracted the disease after inoculation with water in which the mucous membranes of poliomyelitis cadavers had been rinsed. The virus was also found almost constantly in the secretions from the nose and throat of acute poliomyelitis patients. These results are so much at variance with the results obtained by American investigators that the question has arisen whether we are dealing with the same virus in America as that found in Sweden, or whether the virus has marked differences in virulence, or whether there is some experimental error or, more likely, an incorrect interpretation of results.

carriers occur, and are several times more numerous than the frank and abortive cases of poliomyelitis combined. Yet Flexner, Clark and Frazer report only one positive result out of numerous trials. They succeeded in demonstrating the virus of poliomyelitis in the washings from the naso-pharynges of the parents of a child suffering with poliomyelitis. The parents, however, showed no symptoms of illness.

The virus of anterior poliomyelitis is widely diffused throughout the body. It has been found in greatest virulence and concentration in the spinal cord of infected persons and animals. The virus is also quite constantly present in the brain and other organs and tissues as, for instance, the mucous membrane of the nose and pharynx, the mesenteric glands, the axillary and inguinal lymph-nodes, and even in the blood and the cerebrospinal fluid. Recently the virus has been demonstrated in the mucosa of the intestinal tract, and finally in the feces. It is therefore no surprise that a virus with such a wide distribution and so generally diffused throughout the body may occasionally be found in the secretions from the nose and throat. These facts make us hesitate to conclude that infantile paralysis must be a contagious disease, simply because it has been demonstrated experimentally that the virus may be found occasionally in the secretions from the nose and throat; it would be just as logical to conclude that the disease in its spread resembles typhoid fever because the virus has been demonstrated in the mucous membrane of the digestive tube and has also been found in discharges from the intestines.

There are other difficulties which must be met before we can accept as a proved fact that infantile paralysis is a contagious disease. Careful and masterly epidemiologic investigations of poliomyelitis have been conducted by the Massachusetts State Board of Health extending over a period of five years. The results of these studies were summarized by Dr. Mark W. Richardson, who plainly brought out the fact that the disease, as observed in Massachusetts, does not have the earmarks of a contagious disease. The disease prevails in rural rather than under urban conditions. In fact, it shows little tendency to invade cities, and when it does enter the city it does not strike the crowded, congested portions of the city. In all other contagious diseases spread through the secretions from the mouth and nose, epidemic outbreaks have been observed in crowded sections of cities, in asylums, hospitals, jails, on shipboard, and similar places where the spread of infection by contact is favored. This is the case with scarlet fever, diphtheria, measles, mumps, whooping cough, influenza, common colds, pneumonia and finally cerebrospinal meningitis. Cases of infantile paralysis in all stages of the disease have been taken into the hospitals, orphan asylums, children's homes, reform-

atory schools and other institutions in the Commonwealth, but in no instance during the five years in which the disease has been studied has it ever spread under these circumstances.

The seasonal prevalence, furthermore, of infantile paralysis does not suggest the seasonal prevalence of the diseases spread by contact through secretions from the mouth and nose. Almost all such diseases, including cerebrospinal meningitis, occur more particularly during the cold months of the year, whereas the prevalence of infantile paralysis is more marked during the summer months.

The curve of seasonal prevalence of infantile paralysis corresponds more closely with that of typhoid and the diarrhœal diseases than it does with the group of infections spread through the secretions from the mouth and nose. Typhoid has its season of maximum prevalence during the warm weather. Water-borne epidemics are apt to occur in the colder months of the year, and milk outbreaks may take place at any time. "Normal," or residual typhoid is a warm-weather disease and corresponds in this regard with cholera, dysentery and the infantile diarrhœas and other intestinal infections. The only other group of diseases which prevail especially during the warm weather are those which are insect-borne. Yellow fever stops with the first frost. The most pernicious form of malaria (estivo-autumnal) extends into the autumn, but the autumn of tropical regions is warm, rainy and favorable to mosquito life. The season of maximum prevalence of the insect-borne diseases corresponds, of course, to the season of maximum prevalence of insect life, namely, the summer.

It has long been evident to the student of epidemiology that the group of "contagious" diseases spread through the secretions from the mouth and nose occur throughout the entire year, but prevail especially during the colder months. On the other hand, there are two groups of disease having their maximum seasonal prevalence during the warm weather, namely, the intestinal infections and the insect-borne diseases. Of these two groups of summer diseases the insect-borne group disappears almost to the vanishing point in temperate latitudes with wintry climates, whereas the intestinal diseases continue to smolder all winter long, with occasional exacerbations, and sometimes even with outbreaks of epidemic proportions. These are generalizations that may not be applicable to a specific case. When we study the seasonal prevalence of infantile paralysis in all parts of the world, however, we find a summer prevalence, sometimes extending into the fall, but dying down almost out of sight during the winter and spring. So far as we may judge, then, from the seasonal prevalence of this infection, it corresponds more closely with that of the insect-borne type than any other group of diseases.

If poliomyelitis is a contagious disease, then we must construct secondary theories to fit certain known facts in its distribution, seasonal prevalence and age predilection, — facts which are at variance with this theory. The bulk of the cases of poliomyelitis may be very mild, and only those cases, perhaps, are recognized that reach the threshold of clinical observation. A similar situation would be presented if we knew diphtheria only by the cases of postdiphtheritic paralysis. Each case of poliomyelitis, in accordance with the assumption that it is spread by contacts, would be surrounded by a number of healthy carriers, but serious epidemics do not occur because the infected persons are not very susceptible. Why epidemic outbreaks should occur in rural conditions and not in the congested parts of cities is, however, not explained by this assumption. The assumed barrier of resisting individuals apparently isolates the case, but, in fact, favors the spread of the infection. It seems to be a general rule that a region where the disease has been epidemic is spared further outbreaks later. This phenomenon may be explained by assuming that a large part of the population has become immunized by having had the disease in an attenuated and unrecognized form. It is further assumed that in rural districts there is not the same opportunity, perhaps, to acquire immunity, and when an epidemic occurs it is liable to run an exceptionally severe course. The loopholes in these conceptions are evident to students of the disease, but it is an interesting speculation that deserves careful consideration and further study.

That infantile paralysis may be an intestinal infection has not been given the consideration that it deserves. In addition to a suggestive seasonal prevalence there is the age incidence and gastro-intestinal symptoms which often usher in an attack; furthermore, we have the fact that monkeys may be infected by feeding, and the further important fact that the virus has been demonstrated in the intestinal mucosa, and even in the discharges from the bowels. No convincing outbreak of infantile paralysis has ever been associated with water, milk, meat or other article of diet. Furthermore, we would expect a somewhat different epidemiology if food were a medium in transmitting the virus. The inherent unreliability of epidemiologic data, especially of a disease such as infantile paralysis, has already been noted, and the possibility of the virus entering by the digestive tube should be borne in mind by investigators. It took a long time to learn that milk may convey scarlet fever and other infections, and that pork may be responsible for trichinosis.

There has long been a suspicion that man contracts infantile paralysis from the lower animals. Hill incriminates horses. Thus, colts in Minnesota have suffered with a disease clinically like poliomyelitis, and the hypothesis has been proposed that the virus is spread through the intes-

tinal discharges of horses, which, drying, fly about as dust. A number of other students have associated it in one way or another with horses. Joest has described in detail the lesions of a disease of horses known as *Bornasche's Krankheit,* which has a similarity to poliomyelitis. Langhorst considers a possible relationship with the dog and cites two cases, in one of which the patient was bitten by a dog; the other patient was licked by a dog, and at the same time had a few scratches on his hand. In both cases the diagnosis of rabies was not excluded. In fact, there are many striking resemblances between rabies and poliomyelitis. All laboratory investigators who have worked with these two diseases have been struck with this resemblance. Both diseases are acute paralytic affections. The virus in both diseases is found in its greatest concentration and virulence in the central nervous system, but is also widely diffused throughout the body. The virus of both infections is filtrable, and in both affections the brunt of the lesions falls on the neurons of the central nervous system. There are other similarities between the two diseases which should be borne in mind, especially when studying the possible relationship between poliomyelitis and dogs.

Many observations have been made in Massachusetts and elsewhere of paralytic diseases of domestic animals occurring about the same time as poliomyelitis in man. Such paralytic diseases are common among pigs and also chickens, as well as horses, dogs, cats, etc. P. Roemer reported a paralytic disease in guinea-pigs which occurred among animals in his laboratory. The guinea-pigs died of a paralysis which has some resemblance to infantile paralysis. The infection is transmissible from guinea-pig to guinea-pig by inoculation; the virus is found to be non-bacterial and filtrable; the incubation period is from nine to twelve days; the symptoms are flaccid paralysis, usually of the hind legs, with involvement of the bladder. Microscopically, there is also a resemblance in the lesions of these guinea-pigs and those of poliomyelitis in man and monkeys. Roemer, however, does not claim, and there is nothing to indicate, that this paralytic disease of guinea-pigs is identical with poliomyelitis. Neustaedter has recently noted a paralytic affection of guinea-pigs that were kept in a cage with some monkeys with experimental poliomyelitis. The evidence that any of these paralytic diseases were genuine instances of true infantile paralysis is far from convincing.

Animals suffer with many paralytic diseases, the etiology of a few of which are known, but most of which are pathologic puzzles. The mere fact that an animal has an acute paralytic infection, with perhaps suggestive lesions in the cord, is not sufficient basis for concluding that we are dealing with poliomyelitis. All attempts to transmit the virus of

infantile paralysis to lower animals, except the monkey, have failed. Theobald Smith, Flexner and others have made numerous attempts to carry on the paralytic diseases of pigs, chickens and other animals without success. Therefore, while it is fairly possible that some of the lower animals may suffer with poliomyelitis, perhaps in a clinically unrecognized form, and while it is possible that man may contract the infection from lower animals, the possibility is only an assumption and lacks evidence.

Another theory to account for the spread of infantile paralysis is that it is dust-borne. Hill's observations of dust and its relation to the disease in Minnesota have already been referred to. The Massachusetts State Board of Health, during the five years of its epidemiologic studies, also considered the possibility of dust as a medium of conveying the virus, without, however, discovering any particular relationship between dust and the disease. The most suggestive evidence comes from Neustaedter and Thro, who claim to have induced the disease in monkeys by inoculating them with the dust found in sick-rooms. If the virus leaves the body in any considerable amount in the secretions from the mouth and nose, it is quite conceivable that the dust of the sick-room may contain the virus, for we know that, under certain circumstances, it retains its viability for months. Poliomyelitis does not have the characteristics of a dust-borne disease, or even of an air-borne infection, and this hypothesis has therefore been given scant credence.

The possibility that poliomyelitis may be a wound infection has been kept in mind in the investigations made by the Massachusetts State Board of Health. No particular relationship between wounds and the disease has been made out. The resemblance between infantile paralysis and rabies has already been discussed, and the fact is plain that monkeys may be inoculated through wounds; in fact, it is possible to cause the disease in the monkeys in the greatest possible variety of ways.

Some of the reasons for considering poliomyelitis an insect-borne disease have been published in some detail in another publication, and need not now be recounted. The epidemiologic evidence collected by Brues and Shepard, and summarized by Richardson, pointed toward the stable-fly, *Stomoxys calcitrans*. The successful experiments of Rosenau and Brues, soon corroborated by Anderson and Frost, incriminate the stable-fly as a factor in the transfer of the virus. The seasonal prevalence, the rural distribution and other facts concerning the disease are explained on this theory. On the other hand, the experimental facts lack further corroboration, and, moreover, these facts have not been translated from monkey to man, and we are not justified in doing so until further studies,

which are now being made, are available. Schuberg and Kuhn have recently demonstrated that a number and variety of infections may be transmitted by means of the stable-fly. In their experimental work they obtained positive results with relapsing fever, anthrax, southwest African horse sickness (*Pferdesterbe*) and epithelioma of fowls (*Hühnepocken*).

Howard and Clark conducted a series of experiments on insect transmission with the virus of poliomyelitis, at the Rockefeller Institute, with very interesting results. It was found that the domestic fly, *Musca domestica,* can carry the virus of poliomyelitis in an active state for several days on the surface of the body, and for several hours within the gastro-intestinal tract. These experiments were made by permitting the flies to feed on the virus, then killing the insects, grinding up their bodies, filtering, and injecting the filtrate into monkeys. Howard and Clark found that mosquitoes (*Culex pipiens, C. sollicitans and C. cantator*) did not take up and maintain in a living state the virus from the spinal cords of monkeys. Negative results were also obtained with lice (*Pediculus capitis* and *P. vestimenti*). The experiments with lice were designed to simulate natural conditions, but it was found that these insects did not take the virus of the blood of monkeys or maintain it in a living state. The bedbug (*Cimex lectularius*), however, gave positive results in that it was found in one experiment to have taken the virus with the blood from infected monkeys and maintained it in a living state within the body for a period of seven days. When we consider that the virus exists in the blood of monkeys in a very dilute state, for it requires a number of cubic centimeters of blood to infect another monkey, we are almost driven to the conclusion that the virus must have become concentrated (grown?) in the body of the bedbug. The results of Howard and Clark may therefore assume an enlarged significance.

If infantile paralysis is transmitted in nature largely or mainly through the agency of the stable-fly, this fact would render the suppression of the disease comparatively easy, whereas if the infection is spread largely from person to person through the intervention of carriers and missed cases, the difficulties of the problem will be multiplied manyfold. In the case of cerebrospinal fever it has been shown that carriers are ten times as numerous as the cases; if the conditions are analogous in infantile paralysis, the suppression of the disease will probably have to wait on specific therapy, of either preventive or curative nature.

The health officer impatiently asks: "Is poliomyelitis a contagious disease?" "Is it an insect-borne disease?" "Is it dust-borne?" "Is it contracted from lower animals?" "Is it an alimentary infection?" or "Is it possibly, like typhoid fever, spread by several or all of these various

methods of conveyance?" In the present state of our knowledge a definite answer cannot be made to these important queries, and we must await further work before the health officer can direct his measures to combat infantile paralysis with any assurance of success. Meanwhile the public must be given the benefit of the doubt, and the infection fought along all probable lines.

VII.

TRANSMISSION EXPERIMENTS WITH THE VIRUS OF POLIOMYELITIS. FINDING THE VIRUS IN THE NASAL SECRETION OF A HUMAN CARRIER FOUR MONTHS AFTER THE ACUTE STAGE OF A SECOND ATTACK OF POLIOMYELITIS.[1]

William P. Lucas, M.D., and Robert B. Osgood, M.D., Boston.

Since our note on the finding of the virus of anterior poliomyelitis in the tonsils of recovered monkeys,[2] we have been carrying on similar experimentations with human tonsillar and nasopharyngeal tissues as we have been able to obtain them from time to time. Our results in this we presented to the Boston Society of Medical Sciences late in 1911. Our conclusions were that the results of these experimental studies with the filtrates of human nasopharyngeal tissues, removed at varying periods after the acute attack, may be said to be suggestive but not conclusive for the following reasons:

In three monkeys injected with three different tonsillar extracts no clinical signs of poliomyelitis followed the inoculations. In two monkeys the clinical signs were typical, but we were unable to pass the infection on to a second series of monkeys in a perfectly conclusive manner; nor were the pathologic findings absolutely typical, though suggestive, although they were much the same findings as those described by Kling, Pettersson and Wernstedt[3] in their investigations as to the duration of the virus in the human body.

We felt convinced that the nasopharyngeal tissue of recovered human patients was able to retain the virus for at least six months; but owing to the far-reaching importance of such a fact, we withheld the data from print. Since then, however, these facts have been proved conclusively by Kling, Pettersson and Wernstedt, and by Flexner. Further, not only have these investigations proved the presence of the virus in the washings from the nasopharynx of patients in the acute stage of the disease; but also, which is still more significant, the virus has been found in washings

[1] From the Laboratory of Surgical Research, Harvard Medical School. Experiments carried out by means of a grant from the Massachusetts State Board of Health. Reprinted from the Journal of the American Medical Association, May 24, 1913, Vol. LX., pp. 1611, 1612.

[2] Osgood and Lucas: Transmission Experiments with the Virus of Poliomyelitis, the Journal A. M. A., Feb. 18, 1911, p. 495.

[3] Kling, Pettersson and Wernstedt: Investigations on Infantile Paralysis, Report from the State Medical Institute of Sweden to the Fifteenth International Congress on Hygiene and Demography, Washington, 1912.

from the nasopharynx of parents, attendants and friends; and, further, Kling, Pettersson and Wernstedt have found the virus present in nasopharyngeal swabbings and washings as late as 204 days (seven months) after the infection.

The case we wish to report falls into this latter class of carriers, but the probable duration of the carrier was two years and three months.

R. H. (patient of H. G. Rockwell, Amherst, Mass.), aged 5, had his attack of paralysis in February, 1910. He was first seen by one of us (R. B. O.) Feb. 19, 1912. At that time he showed a residual paralysis of the dorsal flexors of both feet and of the peroneal group on the right. He improved till September, 1912; when, after an attack of apparent bronchitis and coryza, he became suddenly weak in the right arm, with increased weakness of the affected legs as well. This was associated with fever, and the mother considered it to be a second attack of paralysis. The boy gradually recovered. Within two weeks after this apparent exacerbation, his little sister had high fever, and one of her arms became completely paralyzed, with weakness of the legs. When the boy was seen on Nov. 8, 1912, six weeks after his recovery from the second attack, he was almost back in muscular strength to where he had been before, without any remaining paralysis of his arm. He still had a certain excess of nasal secretion. Through the efforts of his parents we were able to obtain 10 c.c. of clear mucoid nasal secretion on November 11. With this material passed through a Berkefeld filter, we inoculated two monkeys on November 15, with negative results. Again on Jan. 3, 1913, we received some nasal secretion and inoculated two monkeys with negative results.

We repeated this again, on January 22, injecting 10 c.c. of the filtrate intracerebrally into Monkey 157. This monkey became paralyzed February 2 and died February 5. He developed complete paralysis of all four extremities. The pathologic picture of his cord (Dr. H. C. Low) was not typical but very suggestive.

January 31 we again repeated the injection of filtrate from a fresh specimen of nasal secretion, inoculating two monkeys, Nos. 159 and 160. Monkey 159 received 5 c.c. of the filtrate intracerebrally and 10 c.c. intraperitoneally. He became paralyzed February 5 and died, completely paralyzed, February 8.

Cord and medulla pathologically (Dr. H. C. Low) were typical of anterior poliomyelitis (see appended report); and April 9 we injected two monkeys, one with the cord emulsion of Monkey 157, and the second with the cord of Monkey 159. This last monkey, No. 161, received 8 c.c. of a cord emulsion from Monkey 159. He became paralyzed April 26, and died April 27, with typical complete paralysis. The pathologic findings (see appended report, Dr. H. C. Low) were typical of anterior poliomyelitis.

This case seems to us of special interest; first, as demonstrating the long period during which the virus was undoubtedly harbored in the nasopharynx; secondly, a human infection (his sister) occurring two years after his primary

attack during what seemed like an exacerbation of his former attack; and, thirdly, the recovery of the virus from his nasal secretions four months after his second attack and two years and three months after his first attack; fourthly, the fact that the successful inoculations were done with filtrates from straight nasal secretions, not from washings.

PATHOLOGIC REPORT BY DR. H. C. LOW.

Monkey 159. — Sections of the cord in the lumbar and cervical regions show moderate injection of the blood-vessels and some small round-cell infiltration about them. Changes are not marked but are consistent with the diagnosis of poliomyelitis.

Monkey 161. — Sections of the lumbar, dorsal and cervical regions show marked round-cell infiltration about the blood-vessels and in the perivascular spaces, more evident near the central canal and the anterior horns. Changes are typical of poliomyelitis.

VIII.

EXPERIMENTS TO DETERMINE IF PARALYZED DOMESTIC ANIMALS AND THOSE ASSOCIATED WITH CASES OF INFANTILE PARALYSIS MAY TRANSMIT THIS DISEASE.[1]

By Carl Ten Broeck, M.D., with an Introductory Note by Prof. Theobald Smith, M.D. (From the Department of Comparative Pathology, Harvard University Medical School, Boston.)

Introduction.

The sporadic occurrence of poliomyelitis in numerous, apparently unrelated foci, and the tendency of the disease to appear in rural districts which have only slight intercourse with large centers of population, have led to the hypothesis of some animal reservoir for the virus. The incidence of epidemics in summer, when all animal life is most active, favors this hypothesis. On the other hand, poliomyelitis is inoculable only into monkeys and possibly into rabbits. Other species have shown themselves refractory. Even monkeys are not easily infected, and rabbits are as a rule so difficult to infect that their susceptibility is a matter of debate.

There still remains the hypothesis that certain animals may be carriers of the infection without becoming diseased. The infection may vegetate on mucous membranes without invading the central nervous system.

Hypotheses of this sort do not, as a rule, lead anywhere unless as guides to actual experiments which serve to test their validity. The material available for experimentation under the above hypotheses is so abundant that in planning some investigations to trace the virus of poliomyelitis into the lower animals we thought it best to begin with cases of paralysis not explainable as the result of injury, poisons or well-known infectious agents. Before detailing these experiments a brief survey of what is known of animal diseases simulating infantile paralysis will be in order.

There are several spontaneous diseases of animals associated with paralysis that have strong resemblances to infantile paralysis. P. H. Römer[2] discovered a disease among guinea-pigs which causes paralysis and death. It is due to a filterable agent and may be transmitted from

[1] The cost of this investigation was met by a fund generously contributed by the following gentlemen: Messrs. Frederick S. Converse, William H. Hill, Charles C. Jackson, Charles H. W. Foster, Moses Williams, Moses Williams, Jr., Charles Jackson, Robert Treat Paine, 2d, Frederick P. Royce, Francis R. Bangs, and "A Friend."

[2] Deutsche Med. Wochenschr., XXXVII., 1911, p. 1209.

guinea-pig to guinea-pig by the intracerebral method of inoculation. It is a meningo-myelo-encephalitis with lymphocytic infiltration.

M'Gowan and Rettie[1] describe a poliomyelitis in sheep known as "loupin ill," trembling, etc. This disease begins with fever, restlessness, excitability, trembling and muscular twitching, followed by coma and death or by paralysis producing various deformities and often complete loss of the use of the hind quarters.

The lesions of the central nervous system vary according as the animal suffered from the acute or the chronic type of the disease. In the latter, the pia, nerve roots, the gray and white matter and the perivascular sheaths are infiltrated with small round cells. The nerve cells, especially of the anterior horns of the cord, are in various stages of disintegration. Cultures were negative. Intracerebral inoculations of two sheep with brain tissue from acute cases produced no effect.

In the spinal cord and ganglia of a dog affected with paralysis, Flexner and Clark[2] found lesions closely resembling, but not identical with, poliomyelitis lesions as they are found in man and in inoculated monkeys. The lesions, most pronounced in the cervical and lumbar enlargements, consisted of perivascular infiltrations, hæmorrhage, œdema, infiltration and necrosis of the ground substance and necrosis of ganglion cells, which become at times replaced by small round cells.

Inoculation of two dogs and two monkeys (*Macacus rhesus*) led to no positive result. The animals were under observation for several months.

An infectious disease of horses affecting the central nervous system, which may appear both in sporadic and epidemic form and which has been investigated in Saxony, where it has been prevalent for some twenty years, has recently been studied histologically by E. Joest.[3] This author finds much resemblance between the lesions of poliomyelitis and of this horse disease. The disease is at its height in the spring of the year, and its infectiousness is very slight. The affected animal becomes dull and listless, but the paralyses are incomplete.

Joest finds that the pathological changes are chiefly in the brain. There is a marked lymphocytic infiltration of the perivascular lymph spaces, with a tendency to invasion of the nervous tissue. The spinal cord is much less involved.

Although a diplo-streptococcus has been described by several investigators as associated with the lesions, the etiology is not cleared up. Joest describes intranuclear bodies, and is inclined to regard them as belong-

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[1] Jour. Pathol. and Bacteriol., XVIII., 1913, p. 47.
[2] Jour. Exper. Med., XVII., 1913, p. 577.
[3] Handbuch d. path. Mikroorganismen, 2d edition, Vol. VI., p. 251.

ing to the chlamydozoa of von Prowazek and to contain the virus of the disease.

Attention has been called to the occurrence of paralysis among domestic animals during epidemics or to individual paralyzed animals that have come in contact with cases in families.

Ed. Müller[1] calls attention to the statements which have appeared in medical writings concerning the possible relation between animals and infantile paralysis (Wickman, Krause, Wilke) and adds one case of his own. A child two years old had carried about and played with a paralyzed fowl. No occurrence pointing to contact either direct or indirect with another case could be discovered.

Bruno[2] gives details of two cases occurring in two children of the same family aged two and three years, respectively. The children had not come in contact with other children, but were restricted to a large garden containing poultry and a few sheep. About six weeks before the disease appeared in the children, the father had purchased a number of ducks from an establishment handling many thousands yearly. Five of the ducks became paralyzed, one died, one was killed and three recovered. Bruno does not hesitate to bring this disease of ducks into etiological relation with the cases of infantile paralysis.

Lust and Rosenberg[3] in studying an epidemic in and around Heidelberg fixed their attention upon paralysis in domestic fowls, which was quite common. They inoculated fowls with a suspension of brain and cord of a paralyzed fowl without success. Four young chickens were placed in a hospital ward with cases of poliomyelitis and fed with nasal secretion of such cases for nine days. This experiment also proved negative. Finally, they injected a strain of poliomyelitis virus into nine chickens, either into the brain or into the peritoneal cavity. This experiment likewise failed to produce the disease.

Neustaedter[4] states that two guinea-pigs contracted poliomyelitis by being kept in proximity to a severe case of inoculation poliomyelitis in a monkey. He also found that in one case swabbing the nasal mucosa with filtered virus from the cord of a monkey caused poliomyelitis in a guinea-pig. No subsequent confirmatory experiments have been reported.

The inoculability of the human virus into the lower animals has received considerable attention. Thus far, the tailed monkeys present the most reliable reaction to this virus after intracerebral inoculation. Rabbits have been tried by various experimenters with uncertain and equivocal

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[1] Die spinale Kinderlähmung, Berlin, 1910.
[2] Münch. Med. Wochenschr., 60, 1913, p. 1995.
[3] Münch. Med. Wochenschr., LXI., 1914, p. 120.
[4] Jour. A.M.A., LX., 1913, p. 982.

results. H. K. Marks[1] has more recently gone over this problem again. He found that by using young rabbits, the virus at times may be transmitted through a short series of rabbits and be detected in the last of the series by the use of monkeys. In the experimental work described below, only monkeys were employed, because no other species could be relied upon to yield trustworthy results.

Experimental Part.

Method. — After having been kept under observation for some days the animals were killed, at first, by chloroform, later, as we did not know the effect of chloroform on the virus of poliomyelitis if such should be present on the mucous membranes, by a blow on the head followed by bleeding. A careful autopsy was made and portions of the cord and nasal mucosa removed for inoculation material. In the majority of cases the nasal mucosa was used for inoculation, as it has been shown that in the monkey, at least, the virus persists here long after it can be demonstrated in the cord.[2] We assumed that if the animals were to transmit the disease, they would be more apt to do so through the nose than in any other way that we know of at present. In some cases, however, both nasal mucosa and cord were used for inoculation.

The tissue was ground with sterile sand in a sterile mortar and suspended in salt solution. After standing in the refrigerator over night this suspension was passed through a sterile Berkefeld filter and one or two monkeys inoculated with the filtrate.

Using aseptic precautions and having the monkey under ether anæsthesia, we injected 4 to 6 cubic centimeters into the lateral ventricle. In a few cases we partially sterilized the suspension to be injected, by means of 0.5 per cent. phenol instead of passing it through a filter. The monkeys were kept under observation for at least two months and then, if they remained well, inoculated with tissue from another case, care being taken that it was from a different species than that of the first inoculation, to prevent anaphylactic reaction. In no case was a monkey used more than twice. We lost several monkeys from various causes, as will be seen in the notes, but in all except one case, one of the pair, inoculated from any one animal, lived throughout the incubation period of poliomyelitis. The monkeys used were *Macacus rhesus,* except Nos. 16, 17 and 19, which agreed most nearly with the species description of *Cereocebus galeritus.* The latter were susceptible to poliomyelitis, as we proved by inoculation.

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[1] Jour. Exper. Med., XIV., 1911, p. 116.

[2] Flexner and Clark, Jour. Amer. Med. Assoc., 1911, Vol. LVI., p. 585.

In order to show that by our methods we could produce the disease, we made the two following experiments as controls: —

R. L. — Boy, age 4, had a rise in temperature, pulse and respiration, with vomiting. The next day there was a complete paralysis of the left leg, and the knee jerks were absent. On the fifth day he died, showing at this time complete flaccid paralysis of both legs, left arm and partial paralysis of the right arm, marked difficulty in swallowing. Coarse bubbling rales throughout front and back of both lungs. Cord removed and sent to the State Board of Health, July 21, 1911.

The membranes of the cord showed an intense congestion in the lumbar region, and the vessels of the white matter are well marked. Near the cauda equina there is an intense congestion of the anterior horns and of the white matter. Microscopic examination shows the gray matter infiltrated with lymphocytes and red blood corpuscles, the normal tissue being almost entirely destroyed. Clinical and pathological diagnosis: acute poliomyelitis.

Cord suspended in salt solution, filtered and injected into the lateral ventricles of monkeys Nos. 5 and 6, July 22, 1911. Seventeen days later monkey No. 5 became paralyzed in the right leg, and the next day it had a flaccid paralysis of both hind legs. Nine days after monkey No. 6 was inoculated it became very nervous, lost its appetite, and two days later had a paresis of the left arm and both legs. Gradually, it recovered the use of its limbs, and two months after the inoculation was practically normal. Killed at this time, the autopsy showed lumbar cord slightly congested, the superficial vessels slightly prominent and the anterior horns somewhat reddened. No abnormality noted in the remainder of the cord or in the brain. Pleural and peritoneal cavities with contents normal. Microscopical examination shows a marked perivascular infiltration with lymphocytes in the lumbar cord and a degeneration of the anterior horn cells. A piece of muscle removed from the axilla showed marked atrophy.

Monkeys Nos. 18 and 24. — Inoculated Dec. 28. 1912, with 1 cubic centimeter of a 5 per cent. suspension of the cord of a monkey that had died from the effects of an inoculation with a strain of poliomyelitis virus received from the Hygienic Laboratory of the United States Public Health Service at Washington, D. C. Twelve days later these monkeys were found totally paralyzed, and died the next day. Microscopic examination of their central nervous systems showed the typical lesions of poliomyelitis.

Dogs.

Dog No. 57.[1] — Springfield, Aug. 19, 1911. Dog struck by an automobile two weeks ago. Spastic paralysis of hind legs with retention of feces. Autopsy showed a marked lateral curvature of the spine in the upper thoracic

[1] These numbers are running laboratory numbers and have no bearing on the number of cases of paralysis examined.

region with an enlargement of the bodies of the vertebræ. No inoculations made.

Dog No. 58. — From Dr. G., Boston, Sept. 11, 1911. History of paralysis. Dead when received. Slight indication of injections of minute vessels of pia. Post-mortem: putrefaction well under way.

Filtered suspension of cord injected into lateral ventricle of monkey No. 21. Death in a month from a colitis. No evidence of paralysis. Filtered suspension of nasal mucosa injected into lateral ventricle of monkey No. 22. No effects from inoculation after two months.

Dog No. 59. — North Andover, Sept. 22, 1911. Dog had been sick for about a month. Did not use left fore foot. There seemed to be a hyperæsthesia of all limbs. Chloroformed. Autopsy. Walls of stomach and small intestine thickened and congested. Cord apparently normal except in the lower cervical and thoracic regions, where the gray matter appears to be softer than normal. Inoculations not made as there was no apparent connection between this animal and any cases of poliomyelitis.

Dog No. 60. — West Harwich, Feb. 7, 1912. Spastic paralysis of hind legs with retention of urine and feces. First noticed ten days ago. Autopsy. About 10 centimeters above the lumbar enlargement of the cord for a space of about 3 centimeters the cord was found to be slightly larger, firmer and of a more pearly color than the adjacent tissue. On section a grayish white, pearly mass of tissue was seen replacing the gray matter and most of the white matter of the cord, leaving only a ring of the latter around the periphery. Microscopical examination showed that this tumor was probably a fibroma. No inoculations made as the paralysis was most probably due to the tumor.

Cattle.

Cattle No. 237. — Fairhaven, Nov. 8, 1911. Cow, six years old. Raised on the farm and was a great pet of the children. No cases of poliomyelitis for some years in Fairhaven. Two weeks ago it was noticed that there was a slight incoördination in the cow's movements. This increased until there was a complete paralysis of the hind quarters. Killed and autopsied by M. J. Curran, M.D.V., New Bedford, who reports ascites of the abdominal cavity, organs normal, and no signs of tuberculosis. Cord removed and sent to the State Board of Health.

Cord shows slight congestion. Suspended in salt solution, filtered and injected into the lateral ventricles of monkeys Nos. 18 and 22. Monkey No. 22 became blind and was killed a month and a half after the inoculation. Autopsy showed an internal hydrocephalus. Monkey No. 18 remained well for over two months following the inoculation.

Cattle No. 241. — Sharon. Case obtained by kindness of Dr. Mulvehill, March 1, 1912. Heifer, one year old. Complete motor paralysis of the hind quarters beginning four days ago. No known injury and no evidences of external injury. Autopsy. Distal 15 centimeters of lumbar cord bluish, swollen, with an irregular cavity following the lines of the gray matter and

having a hæmorrhagic border. Microscopical examination of the cord shows no evidences of an inflammatory condition, but does show a diffuse hæmorrhage which has probably softened and produced the cavity. No evidence of injury to spine, but Dr. E. E. Southard, who was consulted, regarded the condition as probably due to trauma. No inoculations made.

Cattle No. 253. — Jan. 8, 1913, from Dr. Langdon Frothingham, three pieces of the fore brain weighing about 20 grams, the medulla and the first portion of the cord.

The brain had been sent in by Dr. Playdon of Reading, Mass., who states that before death the cow had a paralysis of its hind legs and that its head was twisted around as in milk fever. Negri bodies were not found by Dr. Frothingham.

The material was received in a putrid condition, so that microscopic examination was not attempted. The portions of brain and cord were ground in a mortar with sterile sand and suspended in 100 cubic centimeters of salt solution. The suspension was shaken for one hour in a machine, frozen and thawed three times, then centrifuged and the supernatant fluid passed through a Berkefeld filter.

Monkey No. 46 received 4 cubic centimeters of the filtrate into its right lateral ventricle and 42 cubic centimeters into its peritoneal cavity.

Monkey No. 58 received 5 cubic centimeters of the filtrate into its right lateral ventricle.

Monkey No. 46 was under observation for six months following the injection and remained perfectly well during this period. One month after monkey No. 58 had been inoculated, it developed a marked diarrhœa. This disappeared for a time, but reappeared about a month later, and three months after the inoculation the monkey died apparently from this chronic diarrhœa. Microscopical examination of the cord showed normal nerve cells and no perivascular infiltration.

Swine.

Swine, No. 101. — Woburn, Aug. 18, 1911. This pig comes from a neighborhood where there is a case of acute poliomyelitis. Along with four others, this pig was taken sick last winter. The nature of the sickness was not determined, but it was stated that during July there was a similar illness among the pigs, from which over a hundred died. Examination shows that the fore legs are used normally while the hind legs are totally paralyzed. Chloroformed and autopsied. Pig about 100 centimeters long and weighing about 80 to 100 pounds. Viscera in general normal, with the exception of the stomach mucosa, of which the fundus is marked by congestion and is pigmented. Slight erosions around margins of cardiac expansion. No parasites. A portion of the ileum near valve congested. Rectum for a distance of 15 centimeters uniformly distended with dry feces into a cylindrical mass about 3 centimeters in diameter. Urinary bladder well distended into a globular mass equal to two fists put together. Spinal cord at level of lumbar enlargement has a distended vein running along its dorsal aspect; several

similar veins on ventral aspect. No other abnormalities noticed. The vertebræ in this region sawn through, but nothing unusual found. Microscopical examination negative.

Injected the filtered suspension of the nasal mucosa into the lateral ventricles of monkeys Nos. 1 and 2. Monkey No. 1 developed an internal hydrocephalus and died in about a month from the time of the inoculation without showing any signs of paralysis. Monkey No. 2 showed no effect from the inoculation after three months.

Swine No. 105. — North Dana, Feb. 20, 1912. One of five pigs that were taken sick last spring, the others dying. Examination shows a paralysis of the extensor muscles of the hind legs. Given morphine and bled to death. Autopsy showed the cord in the lower lumbar region to be possibly a trifle more moist and softer than normal. Microscopical examination negative.

The suspension of the nasal mucosa partially sterilized by means of 0.5 per cent. phenol, injected into the lateral ventricles of monkeys Nos. 32 and 33. No effect from the inoculation during the following two months.

Swine No. 106. — Westwood, Feb. 8, 1912. This pig came originally from a family where there was a case of acute poliomyelitis. Pig taken with an acute illness and died in twenty-four hours. Unable to use legs, which were hyperæsthetic and somewhat œdematous. Autopsy: white female pig weighing about 75 pounds. Post-mortem: decomposition advanced. Bloody around snout. The membranes of the spinal cord were of a purplish red color, but the individual vessels were not very prominent. In the lumbar region the cord was very soft, and the gray matter could not be distinguished from the white. In the dorsal region the cord was soft, yet the markings were distinct. At no place in the cord was there any apparent congestion. The vessels of the membranes covering the brain were markedly enlarged, but the diffuse reddening of the cord was not present. Peritoneal cavity distended with gas; contains considerable blood-stained fluid. Intestine purplish red in color and distended with gas. Not opened. Spleen not enlarged. Right lung crepitant but firm, not collapsed and dark red in color. Section shows a deeply congested tissue with dark clots in the vessels. Left lung less firm than the right, crepitant and on section of a bright red color.

Frozen sections of the cord showed an exudate of fibrin and polymorphonuclear leucocytes in the meninges, but no perivascular infiltration.

We were not successful in getting the suspension of the nasal mucosa sterile by means of 0.5 per cent. phenol, so that no inoculations were made.

Swine No. 107. — This pig was received May 27, 1912, from Westwood. Three months before a paralyzed pig (No. 106) had been received from the same neighborhood. There was a rather close association with a case of poliomyelitis.

On May 19, 1912, this pig became weak in its fore legs and its hind legs were paralyzed. When received at the laboratory, it was found that the hind legs were in a spastic condition and that the animal could move but could not stand on them. The fore legs were weak. The animal had a rectal

temperature of 107.4 degrees. The pig was killed by a blow on the head. The autopsy showed a rather soft, moist cord, the markings of which were indistinct. The brain was apparently normal except for the congestion due to the blow. The liver, spleen, kidneys and adrenal appeared normal on macroscopic examination. The bladder was distended and the rectum was filled with hard feces. The small intestine was normal. The pancreas was firm and on section numerous bands of a firm, yellowish, soap-like substance were visible. The glands in the groin, under the sternum, at the angles of the jaw and in the mesentery were enlarged. Their cortex was hæmorrhagic, and on section they were light gray in color. The heart and lungs were apparently normal.

Microscopic examination of the tissues of this pig showed an early lymph-node tuberculosis, fat necrosis of the pancreas, together with many focal cell accumulations of lymphocytes under the capsule of the adrenal and around the perilobular veins of the liver. There were numerous, relatively large, focal cell collections in the cortex of the kidneys which crowded upon and compressed the tubules. The cells were of the endothelial type. A few mitoses were seen. Necrosis absent. Most remarkable of all, a very evident perivascular lymphocytic infiltration of the vessels of the lumbar cord. There was no destruction of the nerve cells nor accumulation of lymphocytes in the anterior horns of the gray matter.

Portions of the cord of this pig were placed in 0.5 per cent. phenol and kept in the refrigerator until Nov. 8, 1912 (five and one-third months), when they were washed, suspended in salt solution, and the suspension passed through a Berkefeld filter.[1] Five cubic centimeters of this filtrate were injected into the lateral ventricle of the brain of monkey No. 43 and 50 cubic centimeters into the peritoneal cavity of monkey No. 44. Both animals remained perfectly well throughout the following four months.

Swine No. 114. — This pig was received Oct. 25, 1913, from Framingham. White male pig weighing about 50 pounds. Owner had previously killed one paralyzed pig. This one eats normally. Limbs so weak that animal lies down, but is able to walk to reach his food. Reflexes present. Killed after a week's observation, by cutting vessels of neck after stunning with a blow. All the viscera were normal. There were some fresh small hæmorrhages in muscles of back, probably result of death struggle. Some were found in psoas and muscles of diaphragm. Slight hæmorrhagic infiltration of lymph sinus, lymph nodes of neck, mediastinum, aorta and pelvis. Microscopic examination of muscular tissue fixed and sectioned shows hæmorrhages in the septa between bundles of fibers. Red corpuscles well preserved. No cellular infiltration or other signs of inflammation. The cord of this animal was ground up with sand, suspended in five parts of salt solution by weight, shaken and refrigerated for one day; filtered through

[1] Flexner, Clark and Amoss found that the cord of a child which had died of infantile paralysis, kept for fifteen months in 0.5 per cent. phenol, produced typical paralysis when injected into monkeys (Jour. Exper. Med. XIX., 1914, p. 205).

Berkefeld filter. Filtrate in refrigerator for two days, then injected into monkey No. 63, 2.3 cubic centimeters intracerebral, and 20 cubic centimeters into abdominal cavity. Monkey well after four months.

Fowls.

Fowl No. 25. — Lexington, Aug. 10, 1911. This fowl was received from a family where there is a case of acute poliomyelitis. The only sick chicken in the flock. Stands erect with its sternum pushed out and sways from side to side. Falls over when touched, probably from weakness. No paralysis. Feathers around anus soiled. August 12, dead. Autopsy shows brain and cord normal. Anterior nares plugged with a light yellow fibrinous exudate. Eyes moist but without exudate. Nothing abnormal found in the pleural or peritoneal cavities. Diagnosis: avian diphtheria.

Filtered suspension of nasal mucosa injected into the lateral ventricles of monkeys Nos. 9 and 10. No effect from the inoculations in the following two months.

Fowl No. 26. — Woburn, Aug. 25, 1911. This fowl comes from a flock where two or three chickens are said to have died in a mysterious manner, and where the man who cared for them had an attack of acute poliomyelitis that caused his death. Chicken walks with a limp, as though its foot had been injured. No evident paralysis. Killed by a blow on the head. Autopsy showed fatty degeneration of the liver but no other abnormality.

The filtered suspension of the nasal mucosa was injected into the lateral ventricles of monkeys Nos. 19 and 20, and the filtered suspension of the cord into a lateral ventricle of monkey No. 18. All monkeys remained well during the following two months.

Fowls Nos. 27, 28 and 29. — These three fowls were received Sept. 1, 1911, from Boxford. The principal trouble, as far as their walking went, was an apparent loss of equilibrium. When first received, they would stagger when they walked, but later any movement would cause them to fall forward on their heads. After falling they would lie for some time. There was no evident paralysis. For three years chickens on this farm have had some difficulty in walking which appeared when they were about three months old and which progressed until they died. They have evidently had good care and the owner cannot account for their condition.

Fowl No. 27 was chloroformed September 11, when it was nearly dead. Filtered suspension of nasal mucosa was injected into the lateral ventricle of monkey No. 24; filtered suspension of cord injected into the lateral ventricle of monkey No. 23. Both monkeys remained well during the following three months.

Fowls Nos. 28 and 29 were, on Oct. 24, 1911, bled to death, and the nasal mucosæ of both suspended in salt solution, filtered and injected into the lateral ventricle of monkey No. 17. The brains and cervical cords of the two fowls were suspended in salt solution, filtered and injected into the lateral ventricle of monkey No. 16. The monkey showed no effects from the inocu-

[illegible] during the following two months. Immediately after monkey No. 17 had been inoculated, it was totally paralyzed. A little later it had some convulsions, and the next day it had a left-sided hemiplegia. Two days later it died, evidently from some accident during the operation, the nature of which could not be determined at autopsy.

Fowl No. 30. — Leominster, Sept. 23, 1911. This fowl comes from a flock where there have been several cases of paralysis among the chickens, and where the boy who was intimately associated with them has an acute attack of poliomyelitis. The chicken showed a complete paralysis of the legs, which by October 3 had somewhat improved, but which still prevented it from walking. Bled to death and autopsied. Nothing abnormal found.

Filtered suspension of nasal mucosa injected into the lateral ventricle of monkey No. 26. Well for the following two months. Filtered suspension of cord injected into the lateral ventricle of monkey No. 25. Eight days later the monkey succumbed to an attack of colitis and nephritis.

Fowl No. 40. — Ipswich, March 17, 1914. A very fat adult Plymouth Rock hen. The fowl is unable to stand on its feet apparently on account of a paralysis of the muscles of the back. No atrophy of the muscles can be demonstrated. The toes are flaccid, but are moved by the hen. Both legs seem equally involved. The knee jerks are present and equal. The crossed knee jerks are marked. There is no wing drop and the head is held normally. The comb and feathers are in good condition and no ectoparasites can be demonstrated.

This fowl has not been associated with a case of poliomyelitis and there are no other cases of paralysis among the chickens of this flock, though they have occurred in the flock of the breeder from whom this chicken originally came.

On March 21 the fowl was chloroformed and bled to death. There is no atrophy of the muscles of the back or legs and no evidence of an injury to the spine. The abdominal and thoracic viscera and the central nervous system appear to be normal.

Histological examination of the cord at different levels showed a marked perivascular cell infiltration in the gray matter of the lumbar cord in the pia and, to a slight extent, in the white matter. The same condition was found in sections of the dorsal region, but not so pronounced. None was seen in a section of the cervical region and of the midbrain. The infiltrating cells were of lymphoid type. There was no evidence of neuronophagia, but a small number of the nerve cells were swollen, the Nissl bodies absent, and the cytoplasm very attenuated, almost free from stain. Sections from the cords of fowls Nos. 26 and 27, and one other not referred to above, showed no such changes.

The cord, brain and nasal mucosa were ground with sterile sand and suspended in 75 cubic centimeters of salt solution. The suspension was shaken for one hour and placed in the refrigerator for forty-eight hours. It was then centrifugalized and the supernatant fluid passed through a Berkefeld filter.

On March 24 monkeys Nos. 63 and 64 were each given an intracerebral (intraventricular) injection of 5 cubic centimeters of the filtrate and an intra-abdominal injection of 20 cubic centimeters of the same.

Four days after the inoculation both monkeys were very excitable and had some difficulty in climbing. This lasted for a few days and then gradually passed away, leaving the monkeys apparently normal. At no time was there a definite paralysis. Monkey No. 63 is still normal two months after the inoculation, but monkey No. 64 died suddenly six weeks after the inoculation without showing any paralysis or any other symptoms of note. A very careful autopsy failed to reveal any abnormalities, except slightly congested lungs and a very soft, slightly enlarged spleen. From this last organ a bacterium was obtained which has not been classified, but which is not pathogenic for guinea-pigs. Sections of the cord of this monkey show normal nerve cells and no perivascular infiltration.

Horses.

Horse No. 183. — Aug. 4, 1911. Diphtheria-antitoxin horse has had a drooping of the left eyelid and left side of the lip along with a paralysis of the left fore leg for the last month. Chloroformed. Autopsy showed a blood clot in the Sylvian aqueduct ¼ by 1 inch in size.

The filtered suspension of the nasal mucosa was injected into the lateral ventricles of monkeys Nos. 7 and 8. Both monkeys remained well during the following two months.

Horses Nos. 205, 206 and 208. — These three horses in the same stable in Medford were taken sick in March, 1912, at about the same time with fever, difficulty in swallowing, and a paresis of the legs which made it necessary to suspend them in slings. In Wakefield there was a similar disease among the horses belonging to a man who, along with the owner of the Medford horses, had bought some frozen potatoes, which had been fed in both stables. This was the only known connection between the two places and there was no known association with poliomyelitis. Dr. Playdon of Reading reports that some frostbitten potatoes fed to his rabbits caused similar symptoms, with death in four out of the five animals affected. The disease in the horse was also very fatal, as four of the Wakefield and three of the Medford horses died.

Horse No. 205. — This horse died during the night of March 22–23. Autopsy, March 23, 1912, several hours post-mortem. In the mucosa of the small intestine were found a few hæmorrhagic spots 0.5 centimeter in diameter. The lungs showed a marked pneumonic process on the right side. Nothing else of note was found in the peritoneal or pleural cavities. On opening the skull, considerable cloudy yellow fluid escaped. The vessels of the brain were prominent, and in the region of the left Rolandic fissure over an area 5 centimeters in diameter the cortex was of a decided pinkish hue. Similar areas of less extent were present in other places on the surface of the cerebrum. Nothing abnormal was found in the brain on section. The cord in the region of the fourth, fifth and sixth cervical vertebræ was ap-

y normal. Microscopic examination showed a slight hæmorrhagic
; in the meninges of the brain and cord.
nasal mucosa was treated with 0.5 per cent. phenol, suspended in
ation, and injected into the lateral ventricles of monkeys Nos. 36 and
th monkeys remained well during the following two months.
; *No. 206.* — Autopsy, March 26, 1912, eighteen hours post-mortem.
pneumonia of both lungs. Other viscera normal. Considerable
ained fluid surrounded the brain and the surface of the latter was
d, but nothing abnormal was found on section. The cord in the
f the fifth cervical vertebra showed nothing more than a congestion
membranes. Cultures made from the fluid surrounding the brain
a variety of organisms which were not pathogenic to mice. Micro-
xamination of the central nervous system showed a congestion of
els of the cerebral cortex but no signs of an inflammatory reaction.
ılations were made as the disease was regarded as identical with that
No. 205.
No. 208. — This horse died April 9; autopsy, April 10, 1912. Cord
in packed in ice and sent to the laboratory. The brain showed a
surface congestion, but was otherwise apparently normal. One por-
:he cord was apparently normal, while continuous with it was a region
he membranes were markedly congested and the substance of the
ftened. Microscopic examination of the tissues was unsatisfactory
unt of the decomposition that had taken place before fixation. There
narked hæmorrhage into the membranes but no signs of inflammation.
portions of the cord there was a loss of tissue which involved the
atter, but this was probably due to the poor fixation.
ord was treated with 0.5 per cent. phenol, suspended in salt solution,
ected into the lateral ventricles of monkeys Nos. 38 and 39. Both
s remained well during the following two months.

Cats.

No. 28. — Boston, May 26, 1911. Cat was picked up on the street.
paralysis of both hind legs with retention of urine and feces. Chlo-
ed June 3. Autopsy showed two small hæmorrhagic spots in the
region of the cord. No apparent injury to the spine. Microscopical
ation negative.
red suspension of nasal mucosa injected into lateral ventricle of
No. 1. Filtered suspension of cord and medulla injected into lateral
e of monkey No. 2. Both monkeys appeared perfectly well during
owing two and a half months.
No. 29. — Fitchburg. On June 12, 1911, the cat was noticed to favor
fore leg, and some heat and tenderness were found below the elbow.
ne 16 he had a fit and on examination it was found that he could
either fore leg.
ived at the laboratory Aug. 12, 1911. Examination showed a paralysis
extensor muscles of both fore legs so that they were sharply flexed

at the elbow. Cat ate and felt well during the time it was under observation. August 23, no change in its condition. Killed by a blow on the head. The autopsy showed gray matter of cord hæmorrhagic in the cervical region. (This may have been due to the blow on the head.) Elbow joints freely movable and apparently normal. No visible atrophy of muscles.

Filtered suspension of nasal mucosa injected into the lateral ventricles of monkeys Nos. 16 and 17. Neither monkey showed any effects from the inoculation during the following two months.

Cat No. 31. — Oct. 16, 1911, from Dr. M. F. Hoar of Fall River. Cat was well but had been associated with a case of acute poliomyelitis in a child. Killed by a blow on the head.

Filtered suspension of the nasal mucosa injected into the lateral ventricles of monkeys Nos. 7 and 8. Monkey No. 8 remained well for over two months following the inoculation. A month after monkey No. 7 was inoculated it became blind, but showed no paralysis. Autopsy showed a marked internal hydrocephalus.

Cats Nos. 32 and 33. — Oct. 16, 1911, from Dr. M. F. Hoar of Fall River. These healthy cats came from a family where there was a case of acute poliomyelitis in a child. Killed by a blow on the head. Filtered suspension of the nasal mucosa injected into the lateral ventricles of monkeys Nos. 9 and 10. Monkey No. 9 remained well for over two months following the inoculation. Nearly a month after monkey No. 10 was inoculated, he stopped eating and in another two weeks was very weak, but showed no signs of any paralysis. Autopsy showed a thickening of the walls of the colon with an enlargement of the mesenteric glands. Central nervous system normal.

Cat No. 34. — Chelmsford, March 28, 1912. Cat has been the pet of a child who is affected with acute poliomyelitis. Shows no signs of disease. Killed by a blow on the head. Suspension of nasal mucosa treated with 0.5 per cent. phenol, injected into the lateral ventricles of monkeys Nos. 34 and 35. Monkey No. 34 was well for over a month and then died from an attack of colitis. Monkey No. 35 was well during the following two months.

Cat No. 35. — Amherst, Oct. 5, 1912. This animal was sent to the laboratory with the history that it had had a bad cough and that it had been intimately associated with two children who were suffering from acute poliomyelitis. As the cat had been introduced into the family about three weeks before the onset of the disease in the children there seemed to be a possibility that it might have brought the infection to them.

The cat was under observation in this laboratory for a month and showed no symptoms of paralysis, cough or loss of appetite. It was killed by a blow on the head and the organs appeared normal on gross and microscopical examination.

The nasal mucosa was placed in 0.5 per cent. phenol for twenty-four hours, washed, ground with sand, and suspended in sterile salt solution. After standing over night in the refrigerator, the suspension was passed through a sterile Berkefeld filter.

Five cubic centimeters of this filtrate were injected into the lateral ventricle

of monkey No. 47. The monkey was under observation for ten months and remained perfectly well throughout this period.

Forty-five cubic centimeters of the filtrate were injected into the peritoneal cavity of monkey No. 48. This animal remained well for two months and then developed a diarrhœa and died seventy days after the inoculation from an acute colitis.

Cat No. 36. — Amherst, Nov. 6, 1912. This animal comes from a house about one-quarter mile away from the one where cat No. 35 lived and where there were two cases of poliomyelitis.

This cat was not a pet and spent most of its time around the barn. For the past few months the animal has appeared ill, but has been around the place as usual. One week before it was shipped to the laboratory, a paralysis of the hind legs was noticed.

The animal was received late in the evening and the next morning was found dead, so that no clinical observations were made on it in this laboratory.

Autopsy shows an extensive pneumonia accompanied by a purulent exudate from the nose. The abdominal viscera are apparently normal. The brain and cord on gross examination are normal except in the thoracic region, where the cord appears to be softer and more moist than normal.

Microscopical examination shows an acute bronchopneumonia and an infiltration of lymphocytes in the meninges surrounding the cord.[1]

Portions of the brain and cord of this cat were placed in 50 per cent. glycerine over night, washed, ground up with sand, and suspended in salt solution. This suspension was kept in the refrigerator for three days and then passed through a sterile Berkefeld filter.

Five cubic centimeters of this filtrate were injected into the lateral ventricle of monkey No. 45 and 50 cubic centimeters into the peritoneal cavity of monkey No. 46.

Eight days after the inoculation monkey No. 45 died from an acute colitis, but monkey No. 46 was under observation for a year and failed to show any signs of paralysis or other disturbances.

Cat No. 37. — This cat was brought to the laboratory Dec. 9, 1912, by Dr. S. of Cambridge, who stated that about three weeks ago one of three cats belonging to his family became listless and refused to eat. A few days later it lost the use of its hind legs, dragging them behind it as it walked. This lasted for about three days, after which time it gradually recovered the use of its legs and is now apparently well. The fore legs were not affected and there were no gastro-intestinal disturbances. About eight days from the onset of the disturbance in the first cat, the second cat began to be listless and showed the same symptoms of paralysis as the first one. The cat brought to the laboratory is the third one belonging to the family, and it is supposed to be in the first stages of the disease.

Examination shows an adult yellow and white castrated cat in good con-

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[1] From later studies made on cats it seems probable that this animal was suffering from an infection with bacillus bronchisepticus. In dogs an infection with this organism may be followed by a paralysis, but we have not observed this in cats.

dition. The animal eats very little and remains quiet in its cage. When placed on the floor, it runs and leaps in an apparently normal manner.

On December 12 the cat appears to be stupefied and refuses to eat. When placed on the floor, it moves in a normal manner. Jan. 4, 1913, the cat is perfectly well again. On this date it was killed by a blow and autopsied. Nothing abnormal found beyond a moderate impaction of the rectum with dry feces and a slight distension of the urinary bladder.

The nasal mucosa of cat No. 37 was removed, ground with sterile sand and suspended in sterile salt solution. After standing in the refrigerator for three days, this suspension was filtered and 5 cubic centimeters of the filtrate injected into the lateral ventricle of monkeys Nos. 55 and 56. Monkey No. 55 received in addition 28 cubic centimeters of the filtrate into its peritoneal cavity. Both monkeys made a good recovery from the ether, but monkey No. 55 died twenty-three days and monkey No. 56 sixteen days after the inoculation. Both showed a generalized tuberculosis and neither showed signs of a paralysis between the time of inoculation and the time of death.

As the symptoms shown by cat No. 37 seemed to be of some significance, another cat was placed with it in the same cage. This second animal was under observation forty days and remained well during this period.

Rats.

Two rats were trapped in a house in Waltham in which a case of poliomyelitis had occurred. One of them died and was in a state of decomposition when received at the laboratory. The other was well.

The nasal mucosa, tongue, brain and cord, heart, spleen, kidney, bladder and portions of the liver, lung and rectum with contents from both rats were ground with sand, suspended in salt solution, shaken for two hours, and placed in the refrigerator. After standing for seven days the suspension was filtered through a Berkefeld filter, tested for sterility, and 6 cubic centimeters injected into the lateral ventricles of monkeys Nos. 61 and 62. The latter also received 30 cubic centimeters into the peritoneal cavity. Both monkeys made a good recovery from the ether and beyond a slight attack of diarrhœa remained well during the following five months that they were under observation.

On Nov. 8, 1913, two barn rats were brought to the laboratory with the message that they had been caught in a house in Worcester where there was a case of poliomyelitis. One rat was dead and on autopsy appeared normal.

The second rat sat "hunched" up in the corner of the cage and was evidently very sick. Chloroformed and autopsied. All four feet were very much swollen as a result of œdema. The viscera appeared normal.

Portions of the central nervous system, nasal mucosa, tongue, heart, lungs, spleen, liver, kidney, large intestine with contents and the urinary bladder of both rats were ground with sterile sand, suspended in 100 cubic centimeters of salt solution, shaken for three hours, and allowed to stand in the refrigerator for six days. The suspension was filtered through a Berkefeld filter, tested for sterility and 2½ cubic centimeters injected into the lateral

ventricles of monkeys Nos. 65 and 66. Monkey No. 66 received in addition cubic centimeters of the filtrate into the peritoneal cavity.

Both monkeys showed a good ether recovery, but three hours after the inoculation were very sick, lying on the floor of their cage completely prostrated. For the next three days the monkeys refused to eat and were very sick, but after this time, they gradually improved. During the following three and a half months that they were under observation, they appeared perfectly well.

Flies.

Nineteen specimens of *Stomoxys calcitrans* were caught Nov. 23, 1912, in the antitoxin horse stables of the State Board of Health at Forest Hills. They were ground up with sterile sand and suspended in 40 cubic centimeters of salt solution, placed in refrigerator for twelve days, filtered through a Berkefeld filter and 4 cubic centimeters injected into one lateral ventricle of each of two monkeys (Nos. 51 and 52). No. 52 received also 33 cubic centimeters into the peritoneal cavity. Both monkeys were under observation for four months and remained well.

On Dec. 7, 1912, this experiment was repeated with twenty-two *Stomoxys* obtained from the same stable. The filtrate was prepared as above, with the exception that the suspension remained in the refrigerator but five days. Monkey No. 53 received 5 cubic centimeters of the filtrate into a lateral ventricle and 14 cubic centimeters into the peritoneal cavity. The monkey died in forty-three days as a result of caseous (tubercular) pneumonia and miliary tuberculosis.

SUMMARY.

Besides the animals noted above we have received and autopsied eight fowls, two cats and two cows. Some of these showed evidence of injury to account for their paralysis, some were in a marked state of decomposition, or else were not paralyzed and had not been associated with cases of poliomyelitis, so that they were not used for inoculation.

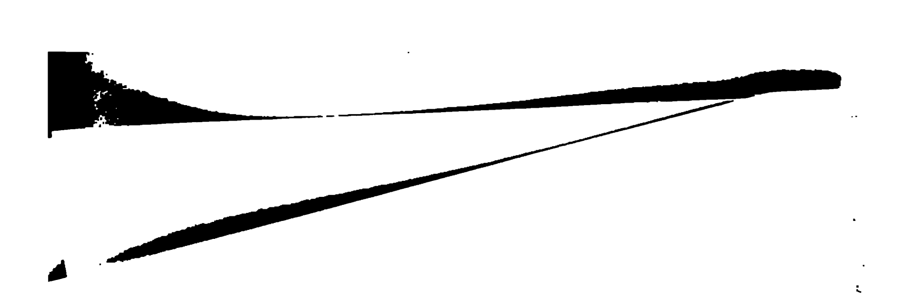
We have received in all forty-eight animals, and material from thirty of these has been injected into monkeys. Of these thirty animals there were four rats, seven fowls, nine cats, three horses, four swine, one dog and two cows. Fifteen were paralyzed, four had a questionable paralysis, and eleven were free from paralysis. Thirteen of these animals had been more or less closely associated with human cases of poliomyelitis, and in the other seventeen no such association was known. *In no case did the monkeys inoculated from any of these animals show any signs of a paralysis or symptoms which would indicate that they were infected with poliomyelitis.* In the cords of those that died no perivascular infiltration with lymphocytes was found nor was there a degeneration of the cells of the anterior horns.

A summary of the inoculations made and the source of the material used is given in the following table: —

Animals furnishing Material to be injected.			Material used (Suspensions).	Site of Injection.	Amount injected (Cubic Centimeters).	Monkey injected (Number).	Result.
Species and Number.	Condition.	Exposed to Infantile Paralysis.					
Dog No. 58.	Paralyzed.	Not known.	Filtered cord.	Lateral ventricle. Lateral ventricle.	5 5	21 22	Death; acute colitis. No effect.
Cattle No. 237.	Paralyzed.	No.	Filtered cord.	Lateral ventricle. Lateral ventricle.	4 4.5	18 22	No effect. Killed; internal hydrocephalus.
Cattle No. 253.	Paralyzed.	Not known.	Filtered brain and cord.	Lateral ventricle. Peritoneal cavity. Lateral ventricle.	4 } 42 } 5	46 58	No effect. Death in three months; chronic colitis.
Swine No. 101.	Paralyzed.	In neighborhood.	Filtered nasal mucosa.	Lateral ventricle. Lateral ventricle.	4 4	1 2	Death; internal hydrocephalus No effect.
Swine No. 105.	Paralyzed.	Not known.	Nasal mucosa treated with 0.5 per cent. of phenol.	Lateral ventricle. Lateral ventricle.	4 2	32 33	No effect. No effect.
Swine No. 107.	Paralyzed.	Yes.	Cord treated with 0.5 per cent. of phenol for five and one half months, then suspended and filtered.	Lateral ventricle. Peritoneal cavity.	5 50	43 44	No effect. No effect.
Swine No. 114.	Paralyzed(?).	No.	Cord filtered.	Lateral ventricle. Peritoneal cavity.	2.3 } 20 }	63	No effect.
Fowl No. 25.	Avian diphtheria.	Yes.	Filtered nasal mucosa.	Lateral ventricle. Lateral ventricle.	1.5 3	9 10	No effect. No effect.
Fowl No. 26.	Limps but is not paralyzed.	Yes.	Filtered nasal mucosa. Filtered nasal mucosa. Filtered cord.	Lateral ventricle. Lateral ventricle. Lateral ventricle.	5 5 4	19 20 18	No effect. No effect. No effect.

...imals furnishing Material to be Injected.			Materials used (Suspensions).	Site of Injection.	Amount injected (Cubic Centimeters).	Monkey injected (Number).	Result.
...pecies and ...umber.	Condition.	Exposed to Infantile Paralysis.					
...d No. 27.	No evident paralysis.	Not known.	Filtered nasal mucosa.	Lateral ventricle.	5	24	No effect.
			Filtered cord.	Lateral ventricle.	5	23	No effect.
...s Nos. ...and 29.	No evident paralysis.	Not known.	Filtered nasal mucosa.	Lateral ventricle.	4	17	Death two days after inoculation.
			Filtered brain and cord.	Lateral ventricle.	4	16	No effect.
No. 30.	Paralyzed.	Yes.	Filtered cord.	Lateral ventricle.	4	25	Death; acute colitis and nephritis.
			Filtered nasal mucosa.	Lateral ventricle.	4	26	No effect.
No. 40.	Paralyzed.	No.	Filtered suspension of brain cord and nasal mucosa.	Lateral ventricle. Peritoneal cavity.	5 } 20 }	63	Excitable four days after inoculation; recovered.
				Lateral ventricle. Peritoneal cavity.	5 } 20 }	64	Excitable four days after and death six weeks after inoculation.
...e No. ...	Paralyzed.	No.	Filtered nasal mucosa.	Lateral ventricle.	3	7	No effect.
				Lateral ventricle.	4	8	No effect.
...e No. ...	Paralyzed.	Not known.	Nasal mucosa treated with 0.5 per cent. of phenol.	Lateral ventricle.	4	36	No effect.
				Lateral ventricle.	4	37	No effect.
...e No. ...	Paralyzed.	Not known.	Cord treated with 0.5 per cent. of phenol.	Lateral ventricle.	4	38	No effect.
				Lateral ventricle.	4	39	No effect.
No. 28.	Paralyzed.	Not known.	Filtered nasal mucosa.	Lateral ventricle.	4	1	No effect.
			Filtered cord and medulla.	Lateral ventricle.	4	2	No effect.
No. 29.	Paralyzed.	Not known.	Filtered nasal mucosa.	Lateral ventricle.	4	16	No effect.
				Lateral ventricle.	4	17	No effect.
No. 31.	Healthy.	Yes.	Filtered nasal mucosa.	Lateral ventricle.	4	7	Death; internal hydrocephalus.
				Lateral ventricle.	4	8	No effect.

Animals furnishing Material to be injected.			Material used (Suspensions).	Site of Injection.	Amount injected (Cubic Centimeters).	Monkey injected (Number).	
Species and Number.	Condition.	Exposed to Infantile Paralysis.					
Cats Nos. 32 and 33.	Healthy.	Yes.	Filtered nasal mucosa.	Lateral ventricle. Lateral ventricle.	4 4	9 10	N D c li
Cat No. 34.	Healthy.	Yes.	Nasal mucosa treated with 0.5 per cent. of phenol.	Lateral ventricle. Lateral ventricle.	4 4	34 35	D c N
Cat No. 35.	Apparently healthy.	Yes.	Nasal mucosa treated with 0.5 per cent. of phenol, then filtered.	Lateral ventricle. Peritoneal cavity.	5 45	47 48	N D e a l c
Cat No. 36.	Paralyzed.	Indirectly.	Filtered brain and cord.	Lateral ventricle. Peritoneal cavity.	5 50	45 46	D c N
Cat No. 37.	No paralysis.	No.	Filtered nasal mucosa.	Lateral ventricle. Peritoneal cavity. Lateral ventricle.	5 28 5	55 56	D b D b
Rats (Waltham).	No paralysis.	Yes.	Portions of various organs suspended in salt solution and filtered.	Lateral ventricle. Lateral ventricle. Peritoneal cavity.	6 6 30	61 62	N N
Rats (Worcester).	No paralysis.	Yes.	Portions of various organs suspended in salt solution and filtered.	Lateral ventricle. Lateral ventricle. Peritoneal cavity.	2.5 2.5 22	65 66	N N
Flies (Stomoxys calcitrans).	Normal.	No.	Bodies suspended and filtered.	Lateral ventricle. Lateral ventricle. Peritoneal cavity.	4 4 33	51 52	N N
Flies (Stomoxys calcitrans).	Normal.	No.	Bodies suspended and filtered.	Lateral ventricle. Peritoneal cavity.	5 14	53	D fc d er b



IX.

STUDY OF AN EPIDEMIC OF INFANTILE PARALYSIS (ACUTE EPIDEMIC POLIOMYELITIS) OCCURRING IN THE SOUTHERN CONNECTICUT VALLEY DISTRICT DURING THE YEAR 1912 (NOV. 1, 1911, TO NOV. 1, 1912).

By James V. W. Boyd, M.D., State Inspector of Health, Springfield, Mass.

Before taking up in detail the discussion of anterior poliomyelitis as it occurred in Springfield in 1912, it will be well to call attention to one or two facts which have come out as a result of a comparative study of the cases as they occurred in 1911, 1912 and 1913. In the first place, a study of the 1910 map shows that cases occurred well along into the winter; in fact, as late as December. In 1911, however, the curious fact was noted that the few cases that occurred took place in the latter months of the year; that is to say, in September, October and November. Indeed, the impression becomes strong that these late fall cases of 1911 constituted really the beginning of the 1912 epidemic, interrupted as is usually the case by the cold season.

If we are to believe, as many do, that anterior poliomyelitis is spread largely through the presence of the virus in the secretions of the mouths and noses of convalescents and also of healthy carriers, how is the fact to be explained that in the early months of 1911, when such carriers must theoretically have been numerous as the result of the 1910 epidemic, no cases of poliomyelitis occurred until late in the fall of 1911? It would appear, most probably, that during this period of immunity, the virus was undergoing a cycle of development, either in the bodies of human beings or in some animal or insect host. It is certainly not to be believed that the material for human infection was not present in the city of Springfield during the spring of 1911 as well as in the fall.

The tendency to a two-year periodicity in Springfield was certainly marked, as shown by the number of cases reported in 1910, 1911, 1912 and 1913.

In 1912, 73 cases were investigated in the following cities and towns: Springfield, Holyoke, Chicopee, Ware, Westfield, West Springfield, Agawam, Southwick, Three Rivers (Palmer), Longmeadow.

In Springfield the epidemic seemed to start in the Italian quarter.

Numerous peddlers live in this district, and it was ascertained that some of these peddlers had illness among their horses. This fact was investigated but, as will be seen later, the information obtained was not very conclusive.

There were few cases among the colored race, a fact noted also in the 1910 epidemic. Furthermore, there were no cases among the Swedish people, whereas, in 1910, that race was considerably afflicted.

In the 1910 epidemic there were cases in three physicians' families. In 1912, only one such case was noted.

The cases occurring in near-by towns seemed to have little connection with the Springfield epidemic directly or indirectly.

From a careful study of all the facts no definite conclusions can be drawn as to the cause and the spread of the epidemic. As in previous epidemics the abortive cases may have been an important factor in the spread of the disease. Recognizing the fact, moreover, that the infection may enter the body through the nose and throat, the fact is unquestioned that insect life has been unusually abundant in the Connecticut valley during the past few years, so that it is not unreasonable to suppose that some insect may have been a factor in the spread of the disease.

Epidemic of Paralysis among the Birds at the State Hatchery in Wilbraham, Mass.

This hatchery is situated in the town of Wilbraham bounded by Sixteen Acres (Springfield) on the north and west, East Longmeadow on the south and west, and the village of Wilbraham on the east. A large number of pheasants, quail, prairie chickens and wild turkeys are raised on this place. The birds are hatched out by common hens, and, when old enough to leave the hen, are placed in large wire coops. When about six weeks old one wing is cut and the birds are then turned loose on the reservation, which is quite extensive.

From July 1 to Sept. 1, 1912, an epidemic of paralysis affected the young pheasants and some of the quail. These young birds seemed to do well until about six weeks old and while they were in their coops. After they had had a wing cut, however, and had been liberated, it was noticed that in a short time they began to sicken, and within a few days died. During this sickness they all became paralyzed and showed some cerebral disturbances. For instance, some would turn somersaults and do other queer antics.

Upon inquiry it was found that they were fed on maggots, which were grown especially for their use. The flies used were of the blue bottle variety and also the biting stable fly. This kind of food was given to them both before they were liberated and afterwards.

At the time the birds were liberated, to be sure, the berries on a large number of blackberry vines were just turning from red to black, and it was at first thought that perhaps these berries had something to do with the sickness of the birds. The last batch of birds, however, that became ill were kept near the house where there was none of these vines.

At the time these younger birds were dying it was also noticed that quite a number of the pheasants seemed to be in poor condition. Of these older birds about 50 died. These birds acted as if doped, were thin and were manifestly not well. They came from Darien, Conn.

Approximately 500 young birds died, and about 50 older birds. Two of the paralyzed birds sent to the Harvard Medical School died, but nothing positive was found in them to account for the paralysis. Two of the older pheasants were also sent, and with them a dead bird that had been killed by a wild turkey. A report received from Prof. Theobald Smith on these birds states that two of them had avian tuberculosis; as to the other, nothing definite was found in the organs, but presumably it was in the early stages of the same disease.

Contemporary Animal Sickness among the Horses.

Toward the last of May and the early part of June 9 horses owned by ice and vegetable peddlers, also one owned by a butcher, became ill. The horses were kept in an unsanitary barn situated in the rear of a butcher store, in that section of the city, furthermore, in which the epidemic started (Italian quarter).

One of the first horses to become ill was that of the butcher. This horse developed gastrointestinal symptoms of a subacute nature. On some days the horse would appear to be fairly well and would be driven for awhile. It would then become ill and would be unfit for use for several days. Finally, one night it became much worse, was unable to get up, and a few days afterwards died. There was no autopsy. A few days afterwards several of the peddlers' horses had similar symptoms, the most prominent of which was a sort of a general weakness. There was no paralysis and none of the remaining horses died. There was a distinct interval between the times of onset in the various cases.

One peddler's horse was in such poor shape that the owner himself became ashamed to drive it, and let a friend take it to his farm in the country.

These horses were attended by William J. Glasgow, D.V.S., who states that he treated, during the summer, other horses which presented similar symptoms. The information was obtained after the epidemic had ceased, however, so that it was impossible to investigate further the illness among these animals.

The cases in Springfield are located approximately in the same districts as those investigated by Sheppard during the epidemic of 1910. A few of them, however, situated in the Forest Park District, show that the disease had invaded to a certain extent territory which had escaped the previous epidemic. As before stated, the epidemic seemed to start in the Italian quarter.

Although the epidemic did not become active until May, there were two cases in November, 1911, one each in December, 1911, and March, 1912. These cases are shown on the map, numbered 46, 47, 48 and 1, and will be described later.

The incidence of the cases as they occurred, by months, in Springfield during 1910 and 1912 is seen in the following tables:—

Cases in Springfield, 1910.

Month.	Number of Cases.	Deaths.
May,	1	-
June,	15	2
July,	67	15
August,	30	3
September,	6	3
October,	5	1
November,	2	1
	126	25[1]

[1] Or 19.8+ per cent.

Cases in Springfield, 1912.

Month.	Number of Cases.	Deaths.
November (1911),	2	1
December (1911),	1	-
March (1912),	1	-
April,	-	-
May,	5	1
June,	3	-
July,	21	8
August,	10	2
September,	4	-
November,	1	1
	48	13[1]

[1] Or 27.08+ per cent.

Although in 1910 there were 126 cases in Springfield, as compared with 48 in 1912, the mortality was greater in the latter year, being 27 per cent. for 1912 and 19 per cent. for 1910.

The height of the 1910 epidemic occurred in July, as was the case in 1912.

Insect Life.

Insect life has markedly increased during 1910, 1911 and especially in 1912, according to the experience of Prof. George Dimmock, who has made a study of the local conditions, and who states that owing to the heavy spring rains (especially during 1912) many hollows have retained their moisture long enough to become breeding places for mosquitoes.

Many children, of course, have given histories of having been bitten by some insect, in most cases by mosquitoes. A number of adults also complained of being bitten by mosquitoes. The common house fly was very numerous, and at different times during the epidemic *stomoxys calcitrans* was found in or near the affected house.

Contemporary Illness.

As in the 1910 epidemic enterocolitis cases were somewhat more numerous than in other years in Springfield.

The following tables show that in 1910 there were more deaths from la grippe than in 1911 and 1912, but in 1912 cerebro-spinal meningitis was a trifle more fatal: —

	La Grippe.	Cerebro-spinal Meningitis.	Enterocolitis.
1910,	12	3	106
1911,	9	1	86
1912,	3	4	96

Some cases that were undoubtedly poliomyelitis were called meningitis, gastroenteritis, la grippe, etc. There were also a large number of abortive cases, which never came to the attention of the authorities.

Poliomyelitis, Outside of Springfield.

Cases developed in near-by cities and towns, but were not so numerous as in the 1910 epidemic. In fact, some places that were afflicted in 1910 escaped entirely the 1912 epidemic. It is also of interest to note that several of the smaller towns, where the disease was not noted in 1910, also appeared to be free from it in 1912.

The following cases, showing human and animal contact, also insect bites and other interesting data from an etiological point of view, are described below in some detail: —

GROUP 1.

Other Cases in the Family.

Case No. 14 (Springfield). — G. T., male, two years. French. Onset. July 11, 1912; paralysis, July 14, 1912. Father, a teamster. Other children, 11, 9, 8, 6, 4, and (2) years old.

When his brother, age six years, was one year old he had paralysis of both legs. He now has one leg paralyzed (left).

Case No. 44 (Springfield). — A. B., male, twenty-eight years. American. Onset, July 1, 1912; paralysis, July 5, 1912. Clerk in bank. This case proved fatal, and death certificate was signed "meningitis." Upon investigation it was found to be poliomyelitis. His five-weeks-old baby came in direct contact with him for several days and died a short time after with the gastrointestinal type of the disease, preceded by respiratory paralysis. (Case No. 45.)

Case No. 46 (Springfield). — E. B., female, two years, two months. Hebrew. Onset, Nov. 9, 1911; paralysis, Nov. 10, 1911. Father, a printer. Other children, 3 years, and (2 years, 2 months) old.

During the 1910 epidemic the sister, now three years old, had a facial paralysis. It is possible that this sister was a carrier.

Case No. 59 (Agawam). — F. P., female, three years. French. Onset, Aug. 6, 1912; paralysis, Aug. 9, 1912. Father, a molder in brass foundry. Other children (3), and 1 year old.

Her baby sister was ill the last of July with gastrointestinal symptoms, and gives a history of having died with respiratory paralysis. The father had "cholera morbus" a few weeks before, which left him too weak to work, so that he had to go out in the country to recuperate (a possibly abortive case).

Cases Nos. 60 and 61 (Agawam). — R. B., female, nine years. Negro. Onset, July 25, 1912; paralysis, July 29, 1912. Father, a chauffeur. Other children, (12), and (9) years old. Live on a small farm, had been picking strawberries almost daily with her sister, who had an onset July 28, 1912, and developed paralysis July 29, 1912. (Case No. 61.) It is possible that they became infected at the same time. Both of these cases made complete recoveries.

Case No. 70 (Westfield). — J. S., female, two years. American. Onset, Sept. 30, 1912; paralysis, Oct. 6, 1912. Father, a carpenter. Other children, 10, 8, and (2) years old.

A brother had the disease in 1910, both legs being paralyzed. He has since made a complete recovery. The patient was not born at that time.

Cases Nos. 72 and 73 (Longmeadow). — E. H., female, two years. Amer-

ican. Onset, May 17, 1912; paralysis, May 21, 1912. Father, register of deeds. Other children, (2), and (4) years old.

The brother, age four years, became sick May 23, 1912, but the case was an abortive one.

GROUP 2.

Cases occurring in a House where there was a Case in 1910.

Case No. 4 (Springfield). — A. G., male, twenty-two months. Italian. Onset, May 8, 1912; paralysis, May 9, 1912. Father, a chauffeur. Other children, (22 months), 10 days. A boy downstairs had the disease in 1910 and still resides there.

Case No. 10 (Springfield). — I. B., female, two and one-half years. American. Onset, July 4, 1912; paralysis, July 7, 1912. Father, a train despatcher. Only child.

A baby downstairs had the disease in 1910, but no history of contact with the case.

Case No. 34 (Springfield). — C. S., female, seven years. German. Onset, Aug. 2, 1912; paralysis, Aug. 7, 1912. Father, a bookkeeper. Other children, 11, 9, 7, (7), 5 years, and 11 months old.

A girl upstairs had the disease in 1910, and still shows partial paralysis in one extremity. These two children were quite intimate.

GROUP 3.

Cases which had Abrasions, Sores, Punctured Wounds, whereby Infection could enter, Other than through Mucous Membranes or Insect Bites.

Case No. 11 (Springfield). — C. S. C., male, eighteen years. American. Onset, July 9, 1912; paralysis, July 10, 1912. Father, a physician. Other children, (18), 15, 3, and 3 years old.

Some time in June he ran a rusty nail into his foot while tearing down an old church. The wound healed in a short time.

Case No. 43 (Springfield). — G. P., male, fifty-six years. American. Onset, Oct. 17, 1912; paralysis, Oct. 24, 1912. Occupation, machinist. He had a sore on left malar bone; scab came off about a week before he became ill and had not healed over. Gives no history of insect bites.

Case No. 53 (West Springfield). — E. L., male, two years. American. Onset, May 17, 1912; paralysis, May 19, 1912. Father, a laborer on the railroad. Other children, (2), and 12 years old.

This child had a ringworm on his scalp for several weeks before his onset, also a sore on his nose which had been there for several months.

Case No. 64 (Holyoke). — C. T., female, one year, eight months. American. Onset, Aug. 13, 1912; paralysis, Sept. 4, 1912. Father, a patrolman. Other children, 2½ years, and (1 year 8 months) old.

This child was found to be suffering with impetigo contagiosa for a month past; location, face and scalp.

Group 4.

Cases which came in Close Contact with Sick or Suspiciously Sick Animals.

Case No. 15 (Springfield). — M. W., male, one and a half years. French. Onset, July 15, 1912; paralysis, July 17, 1912. Father, a teamster. Other children, 4, and (1½) years old. This child was known to fondle a sick cat owned by a family downstairs. The cat mysteriously disappeared before it could be obtained for investigation.

Case No. 26 (Springfield). — E. A. F., male, one year and four months. American. Father, a chauffeur. Other children, 13, 8, 4 years, and (1 year 4 months) old. This child played with a sick cat owned by a family upstairs. This cat was ill two weeks before the child had his onset. Unfortunately the people killed the cat before it could be obtained for investigation.

Case No. 28 (Springfield). — R. E. B., male, seventeen years. French. Onset, July 27, 1912; paralysis, July 30, 1912. Stock clerk. He was very fond of animals, and is known to have petted a kitten next door. This kitten was suspected of being ill.

Case No. 31 (Indian Orchard, Springfield). — B. K., male, thirty-four years. Scotch. Onset, June 10, 1912; paralysis, July 23, 1912. Occupation, valve tester. This man had a cat which became ill about a week before. The animal was obtained and sent to Harvard Medical School for observation. Nothing positive was found to exist.

Case No. 34 (Springfield). — C. T., female, seven years. German. Onset, Aug. 2, 1912; paralysis, Aug. 7, 1912. Father, a bookkeeper. Other children, 11, 9, 7, (7), 5 years, and 11 months old.

This child played with a kitten which had come from a family where they had a case of poliomyelitis in 1910. The animal was sent to the Harvard Medical School for observation, but nothing positive was found.

Case No. 49 (Ware). — J. G. G., male, thirteen months. American. Onset, Sept. 27, 1912; paralysis, Sept. 28, 1912. Father, a farmer. Other children, 6, 3 years, and (13 months) old. On this farm there were two paralyzed chickens in 1911. Within the last few months a cat had several litters of kittens all of which died with some cerebral symptoms, rigidity of neck and impaired locomotion. The mother cat suddenly disappeared. A horse had tetanus some months previous.

Cases Nos. 72 and 73 (Longmeadow). — Described above under Group 1, these cases came in contact with a sick cat in the family. This animal had been killed, so it was impossible to obtain it for investigation.

Group 5.

Cases which are of Interest for Other Reasons.

Case No. 1 (Springfield). — M. M. G., male, thirty years. Irish. Onset, Feb. 25, 1912; paralysis, Feb. 26, 1912. This man worked in a brewery, washing bottles. He was exposed to a continual dampness. It was first thought that he had rheumatism, and was so treated by several physicians.

Case No. 3 (*Springfield*). — A. F., male, thirteen months. Italian. Onset, May 8, 1912; paralysis, May 9, 1912. Father, a laborer. Other children, 9, 8, 7, 3 years, and (13 months) old. This child developed herpes zoster on the chest and the left shoulder and neck.

Case No. 13 (*Springfield*). — M. F. S., female, seventeen months. American. Onset, July 13, 1912; paralysis, July 14, 1912. Father, an iceman. This child's father is a driver for a large ice company. There was an epidemic of so-called pink eye in the barn where he kept the horses, and one of his horses was ill. It is possible that the father may have transported *stomoxys calcitrans*. This case was investigated by Mr. C. T. Brues, entomologist, but nothing definite was obtained.

Case No. 17 (*Springfield*). — A. W., female, seven years. American. Onset, July 15, 1912; abortive case. Father, an artist. Other children 9, and (7) years old. This was a very interesting case from the fact that it was next door to where there were two fatal cases. If there was any contact between these it was very indirect. The patient had her tonsils removed some ten days before by a specialist, whose son was afflicted with the disease on the 9th of July. This child was very ill and had the typical symptoms of poliomyelitis without the paralysis.

Case No. 18 (*Springfield*). — E. B., female, eighteen years. American. Onset, July 15, 1912; paralysis, July 22, 1912. Father, an attorney-at-law. During the week of July 7 she was on a farm in Worthington, where there were all sorts of domestic animals. There was no history of any illness among them. She also waded in a mountain brook, the water of which was very cold, and came in contact with the grandmother of a child who had the disease in another part of the town. It is not known whether this grandmother came in actual contact with the case.

Case No. 19 (*Springfield*). — M. C. D., female, eight months. French. Onset, July 9, 1912; paralysis, July 16, 1912. Father, a teamster. Other children, 13 years, and (8 months) old. This child came in intimate contact with two cases, which proved fatal. The two fatal cases were cousins of the patient, and the fathers of each of these were teamsters.

Case No. 22 (*Springfield*). — D. B. L., male, one year. American. Onset, July 22, 1912; paralysis, July 26, 1912. Father, a lumberman. The father of this child had been suffering with herpes zoster for the past three weeks. Several weeks before onset the child had been for a long automobile ride in Vermont, and was known to have stopped at Stratton and West Townsend. Living near this child was another case who had the disease in 1910, and who came in contact with him.

Case No. 23 (*Springfield*). — D. D. A., male, four years. American. Onset, July 1, 1912; paralysis, July 27, 1912. Father, a clerk in a railroad office. Other children, 19, 16, and (4) years old. This boy had a herpes of the face and mouth. His four sisters, 16, 19, 21, and 24 years old, also the mother and father, together with a visiting aunt, all had symptoms of mild grippe. This boy afterwards developed the same symptoms and became

paralyzed. It is reasonable to suppose that the rest of the family were abortive cases of the disease.

Case No. 25 (Springfield). — G. W., male, nineteen years. American. Onset, July 20, 1912; paralysis, July 23, 1912. Occupation, clerk and bookkeeper. This young man was very sluggish mentally for several days during his attack, and suddenly developed acute mental excitement, sleeplessness, loss of memory, and at times became violent and had to be restrained.

Case No. 39 (Springfield). — I. O'C., male, two years. Irish. Onset, Aug. 2, 1912; paralysis, Sept. 2, 1912. Father, a laborer for the city. Other children, 12, 10, 5, and (2) years old. This child came in daily contact with another child who developed the disease previously. The other child died.

Case No. 47 (Springfield). — T. J. B., male, nineteen years. Assyrian. Onset, Nov. 7, 1911; paralysis, Nov. 7, 1911. Occupation, barber. He was found to be covered with pediculi, and may have received the infection from the bites of these parasites.

Case No. 48 (Springfield). — L. W., female, four years. American. Onset, Dec. 27, 1911; paralysis, Dec. 29, 1911. Father, a print builder. Other children, 21, 18, 14, 11, and (4) years old. A week before this child's onset her sisters, 14 and 11, had what was thought to be grippe colds. (Possible abortive cases.) This family used only canned milk.

Case No. 51 (West Springfield). — C. J. J., male, six months. American. Onset, July 20, 1912; paralysis, Aug. 3, 1912. Father, a mechanic. Other children, 5, 3 years, and (6 months) old. He was taken ill in West Springfield, but was later taken to Blandford where he died. The child had been in poor health since it was born and had very little resistance.

Case No. 52 (West Springfield). — A. R. B., female, three months. French. Onset, Aug. 4, 1912; paralysis, Aug. 11, 1912. Father, a shipping clerk. Other children, 4, 2 years, and (3 months) old. A week following the death of this child the other two children became ill with fever, vomiting and diarrhœa. The boy, age four years, had convulsions, probably abortive poliomyelitis.

Case No. 58 (Agawam). — H. D. C., male, three years. French. Onset, July 3, 1912; paralysis, July 6, 1912. Father, a harness maker. Other children, 10, 9, 7, 5, and (3) years old.

This child came in immediate contact with another case near his home. The other case had not been diagnosed correctly for several days and exposed quite a number of other people.

Case No. 63 (Three Rivers). — P. M., female, three years. American. Onset, Aug. 7, 1912; paralysis, Aug. 16, 1912. Father, a carpenter. Other children, 9, 5, (3) years, and 10 months old. Child gives a history of being on a train several days before her onset. Across the aisle in the car was a sick child who was constantly vomiting (a possible poliomyelitis case).

Case No. 66 (Southwick). — A. F. W., male, sixteen years. American. Onset, Sept. 19, 1912; paralysis, Sept. 26, 1912. Father, a farmer. Other children, (16), 6 years, and 16 months old. He worked on a farm after he left high school in Westfield (June). In his class at school he came in contact

with a young girl who died with the disease July 26, 1912. He had been visiting in Springfield during the last week in August, but there was no illness in the family at the time. He was first taken ill in Westfield, but immediately removed to his home in Southwick. Although exposed to the *Stomoxys calcitrans L.*, he gives no history of being bitten by them; neither was there any illness of the animals on the farm.

Case No. 69 (*Westfield*). — M. L. S., female, thirty-three years. American. Onset, Sept. 22, 1912; paralysis, Sept. 23, 1912. Occupation, school-teacher. One child in family, 11 years. She taught school in Springfield, and while on a week-end visit to her sister in Westfield was taken ill. Gives a history of an insect bite on her arm about Sept. 13, 1912. No active case of poliomyelitis known among the school children where she taught.

Case No. 71 (*Westfield*). — F. R., female, sixteen years. American. Onset, July 23, 1912; paralysis, July 26, 1912. Father, a farmer in Blandford. She was in Blandford when first taken ill, but went to Westfield and became worse. She gives a history of being bitten by some insect on her left cheek several days before her onset. On the farm where she lived there were numerous stable flies and mosquitoes. No history of any sick animals however.

Detailed Analysis of Cases.

Of the 73 cases investigated, the following is a detailed analysis. The ages varied from under one year to fifty-six years, as shown in the following table: —

	Cases.
Under one year,	7
From one to two years,	16
From two to three years,	14
From three to four years,	8
From four to five years,	7
From five to ten years,	6
From ten to fifteen years,	2
From fifteen to twenty years,	6
From twenty to twenty-five years,	–
From twenty-five to thirty years,	2
From thirty to thirty-five years,	3
From thirty-five to forty years,	–
From forty to forty-five years,	1
From forty-five to fifty years,	–
From fifty to fifty-five years,	–
From fifty-five to sixty years,	1

There were 39 males and 34 females afflicted with the disease, the youngest five weeks, and the oldest fifty-six years.

The cases occurred among the following nationalities, as shown in the table below: —

	Cases.
American,	37
Irish,	5
French,	11
Italian,	6
German,	1
Scotch,	1
English,	1
Hebrew,	4
Polish,	2
Assyrian,	2
Negro,	3

The number of cases in the different cities and towns are as follows: —

	Cases.
Springfield,	48
Holyoke,	1
Chicopee,	1
Westfield,	5
Ware,	2
West Springfield,	6
Longmeadow,	2
Agawam,	5
Three Rivers (Palmer),	2
Southwick,	1

The sanitary conditions are given in the following table: —

Sanitary Conditions.	Cases.
Excellent,	23
Good,	27
Fair,	9
Bad,	14

Families with one positive case,	63
Families with two positive cases,	7

Cases among acquaintances or neighbors,	10
Cases in which there was illness in the family at the time,	3

Prevalent disease in the cities and towns at the time was not of sufficient importance to be noticed, except that gastrointestinal disturbances were more common.

	Cases.
House pets or domestic animals ill in the vicinity,	3

Diet.

	Cases.
General (including meat, fish, fruit, berries, cereals, bread, milk and eggs),	55
General with strawberries,	1
General with nursing,	1
Nursing baby,	5
Crackers and milk,	1
Bread and milk,	1
Cereals and milk,	1
Milk,	7
Nestlés Food,	1
Raw cows' milk used in	60
No specific article of diet,	65
Cereals,	1
General, in small amount of each,	3
General, without fish, small amounts,	1
Fruits,	1
Crackers and potatoes,	1
Bread and crackers,	1

Unusual Article of Diet within Two Weeks of Attack.

Strawberries,	5
Raw rhubarb and strawberries,	1
Cherries and strawberries,	1
Blackberries,	1
Bananas,	1
Bananas and oranges,	1
Bananas and apples,	1
Raw pears,	1
Clam chowder,	1
Little neck clams,	1
Welch rarebit,	1
Green grapes,	1
Ice cream,	3
Apples,	1
Tomatoes,	1
Crackers,	1
As to exposure to heat,	–
As to exposure to cold,	–
As to exposure to dampness,	7
Acute illness within four weeks of onset of disease,	7
Overexertion preceding attack,	4
Accident preceding attack,	4
Fall preceding attack,	7

Condition of Patient previous to Onset of Disease.

	Cases.
Good health,	25
Headache,	1
Sickly,	3
Fretful and teething,	8
Laryngitis,	1
Backache,	2
Drowsy, tired, hard to raise feet,	1
Symptoms of grippe,	1
Mentally dull,	5
Toothache,	1
Fretful and irritable,	3
Herpes of face and mouth,	1
Fretful,	15
Nervous,	1
Quieter than usual,	1
Worried account of pain in chest,	1
Irritable,	2
Nightmare,	2

Appearance of Paralysis after Onset of Fever.

Same day,	4
One day,	17
Two days,	11
Three days,	8
Four days,	10
Five days,	2
Six days,	4
Seven days,	8
Nine days,	1
Ten days,	1
Eleven days,	1
Twelve days,	1
Fourteen days,	1
Three to four weeks,	1
Six to seven weeks,	1
Abortive cases,	2

General Features of Acute Attack.

1. Sixty cases gave a history of fever, as follows: —

100° F.,	31
101° F.,	10
102° F.,	11
103° F.,	5
104° F.,	3

2. Thirty-nine cases showed brain symptoms, as follows: —

	Cases.
Insomnia,	5
Delirium,	11
Headache,	13
Drowsiness,	2
Twitching, crying in sleep,	1
Convulsions,	3
Stupor and dulness,	4

3. Thirty-nine cases showed retraction of the head.
4. Sixty-five cases showed pain or tenderness.
5. The following cases showed unusual symptoms: —

Sore throat,	2
Dulness followed by acute mental excitement, resembling mania,	1
Cough,	1

There were 64 persons affected with digestive disturbances, — (*a*) those accompanying the attack, (*b*) those preceding the attack, and (*c*) those following the attack, as follows: —

	Preceding Attack.	Accompanying Attack.	Following Attack.
Constipation,	7	28	-
Vomiting,	17	29	1
Diarrhœa,	3	9	-
Colic,	2	-	-

Nine cases had no digestive disturbances.

Distribution of Paralysis at its Worst.

The first table shows the totals of these paralyses according to the members of the body affected, and the second table shows the combination of paralyses affecting each individual.

(1) *Distribution of Paralysis.*

	Cases.
Paralysis of right thigh,	38
Paralysis of left thigh,	33
Paralysis of right leg,	39
Paralysis of left leg,	34
Paralysis of right forearm,	17
Paralysis of left forearm,	18
Paralysis of right arm,	18
Paralysis of left arm,	20

	Cases.
Paralysis of back,	22
Paralysis of abdomen,	9
Paralysis of the right face,	1
Paralysis of the left face,	3
Disturbances of the bladder during the attack,	9
Disturbances of the rectum during the attack,	6

(2) *Paralysis, as last seen.*

Right back,	1
Right leg,	2
Left leg,	1
Left leg, both arms,	1
Both legs, left arm and back,	1
Right leg, left face,	1
General except in face,	2
Mostly right deltoid, slight in right biceps and triceps,	1
Right thigh and leg,	5
Both legs,	7
Respiratory,	4
Left thigh and leg,	4
Left arm and forearm,	2
Both legs and back,	3
Abortive,	2
Left leg and back,	1
Deltoid and back,	1
Left leg, arm and back,	1
Both legs and left arm,	1
Left leg and both hands,	1
Back,	2
Legs and hands,	1
Arms,	2
Neck, back, arms and right leg,	1
Right arm, both legs,	2
Right glutial,	1
Legs, back and arms,	2
Deglutition,	1
Legs, arms, back and bladder,	1
Both thighs and legs,	2
Legs, arms, back and abdomen,	1
Abdomen and back,	1
Right arm,	1
Right deltoid,	1
Left leg and throat,	1

	Cases.
Left face and respiratory,	1
Laryngeal,	1
Left face,	1
Left arm, leg and right face,	1
Legs and arms,	4

The duration of the pain or tenderness in the paralyzed cases is as follows: —

	Cases.
A few days,	47
Two weeks to ten days,	3
One week,	3
One month,	1
Three days,	2

The paralysis entirely disappeared in but 3 cases.

Line and effectiveness of treatment show nothing important.

	Cases.
Urotropin,	36
Urotropin and ergot,	1
Phenacetin and salicylates,	1
Palliative,	13
Bromide,	1
Ergot,	1
Strychnine,	1

Of the 19 fatal cases of this disease the symptoms were respiratory paralysis (preceding death).

Approximate Date of Recovery.

	Cases.
Not stated,	45
One week,	2
Few weeks,	5
Few days,	1
Six months,	1
Fatal,	19

There were no autopsies performed.

There was but one case of lumbar puncture in the 73 cases. This showed a clear cerebro-spinal fluid.

There were no blood examinations made.

There was one urine examination made, which proved to be negative.

Occupations of Adults in the Family.

Carpenters,	5
Laborer (washing bottles),	1
Laborer (forge shop),	1
Foundry helpers,	2
Laborer (waste company),	1
Laborer (metal body worker),	1
Laborers,	2
Bridgemen,	2
Chauffeurs,	3
Mason,	1
Stationary firemen,	3
Barbers,	2
Stock-room clerk (auto company),	1
Skate hardener,	1
Train despatcher,	1
Physician,	1
Salesman, soda water,	1
Ice man,	1
Teamsters,	4
Shipping clerk, also ruler of paper,	1
Artist,	1
Lumberman,	1
Clerks, railroad office,	2
School-teachers,	2
Tailor,	1
Attorney,	1
Servant girl,	1
Traveling salesman,	1
Steam fitters,	2
Valve tester,	1
Peddler,	1
Manager rubber company,	1
Bookkeeper,	1
Bartender,	1
Truckman on street railway,	1
Expressman,	1
Assembler autos,	1
Pattern maker,	1
Machinists,	3
Clerks,	2
Printer,	1
Farmers,	3
Boarding-house keeper,	1

Cement worker,	1
Tar roofer,	1
Harness maker,	1
Molder,	1
Patrolman,	1
Preacher,	1
Register of deeds,	1

Among the 73 cases there were 14 with lodgers in the family.

Occupations of the Lodgers.

Hod carriers,	2
Tar-paper roofer,	1
Laborers,	11
Schoolboy,	1
Teamsters,	2
Clerk, paper manufacturing company,	1
Ice men,	2
Chauffeur,	1
Peddler,	1
Housekeeper,	1
Instructors (Y. M. C. A.),	2
Upholsterer,	1
Lithographer,	1
Farm hand,	1
Railroad man,	1
Baker,	1
Machinist,	1

The following table shows the number of children in the 73 cases, according to family: —

	Cases.
Not any children in the family,	8
One child in the family,	14
Two children in the family,	20
Three children in the family,	13
Four children in the family,	8
Five children in the family,	3
Six children in the family,	4
Seven children in the family,	2

There were 179 children in 70 families, their ages being as follows: —

Children under one year,	18
Children from one to five years,	74

Children from five to ten years,	41
Children from ten to fifteen years,	31
Children from fifteen to twenty years,	12
Children from twenty to twenty-five years,	3

Of the 179 children, 83 were males and 96 females.

Number of Other Children who played with the Patient.

Contact with 2 children,	2
Contact with 3 children,	3
Contact with 4 children,	6
Contact with 6 children,	7
Contact with 10 children,	1
Contact with 12 children,	9
Contact with 15 children,	1

Of the remaining cases it might be stated that anywhere from 1 to 25 persons may have come in contact with the patient.

House, Old or New.

	Cases.
House, old,	54
House, new,	19

Length of Time living in House.

A few days,	2
Less than one year,	27
From one to two years,	16
From two to three years,	7
From three to four years,	2
From four to five years,	7
From five to six years,	1
From six to seven years,	3
From seven to eight years,	3
Nine years,	1
Eleven years,	1
All their lives,	3

House situated on High or Low Ground.

On high ground,	33
On low ground,	40

House situated on Dry or Damp Ground.

On dry ground,	42
On damp ground,	31

Relation to Dust.

	Cases.
With dust,	40
Without dust,	33

Road watered or oiled.

Watered and oiled,	12
Watered,	15
Oiled,	23
No treatment,	23

Location of House.

Nearness to railroad: —	
Within a block of the railroad,	22
Within one-half mile of the railroad,	34
Within one mile of the railroad,	13
Over one mile from the railroad,	4
Nearness to streams, ponds, etc.: —	
Within a block of the water,	18
Within one-half mile of the water,	34
Within one mile of the water,	19
Over a mile from the water,	2
Nearness to the highroad: —	
Within a block of the highroad,	18
Within one-half a mile of the highroad,	34
Within one mile of the highroad,	17
Over a mile from the highroad,	4
Nearness to the car line: —	
Within a block of the car line,	10
Within a half-mile of the car line,	34
Within a mile of the car line,	25
Over a mile from the car line,	4

Out of the 73 cases there had been recent illness in 20 families.

Paralysis in family, friends or relatives before or since attack, as shown in the following table: —

	Cases.
Paralysis in sister,	5
Paralysis in brother,	2
Paralysis in cousin,	1
Paralysis in boy, same house, downstairs,	1
Paralysis in neighbor's baby, next street,	1
Paralysis in neighbors,	2
Paralysis in father,	1

Movements of Patient and Family previous to Illness.	Cases.
At home,	36
Trolley rides, Riverside Grove,	17
Trolley rides, public parks,	9
Trolley rides,	7
Railroad,	7
Automobiling in country,	2
Bicycle riding,	1

In 4 cases out of the 73 there had been members of the family visiting.

There was no illness in the places visited.

There were no letters or other articles received from the houses in which sickness existed.

School Attendance.

Schools attended by patients,	3

There were no paralyzed children in the schools.

Habits and Amusements of the Patient.	Cases.
Baby in arms,	17
Baby in carriage,	2
Playing in the house,	1
Playing in the yard,	5
Sickly,	1
Working man,	2
Active,	45

Wading, Swimming or Paddling.

At Forest Park, Watershop Pond, Mountain Park and Worthington,	10
Fell into the water,	1

In 5 of the above cases the water was clean, and in 6 of these cases it was not clean.

In 3 cases out of the 73 there had been digging or excavating in their neighborhood.

Water Supply and Sewage.	Cases.
Good,	61
City water supply and bad water-closet,	2
Well water and privy,	1
Fair,	2
Good water and filthy privy,	1
Privy,	2
Bad privy,	1
Well and privy,	2

Animals or Birds in the House.	Cases.
Cats,	16
Cat and dog,	6
Dog,	3
Chickens,	2
Birds,	2
Dog next door,	1
Cat next door,	1
Horses and cows,	1
Kittens, cows and horse,	1

How recently acquired.	
Several months,	3
Few months,	5
Four weeks,	2
Three weeks,	2
One year,	5
One to two years,	2
Two years,	1
Six years,	4
Four years,	1
Ten years,	1
Three years,	2

In 7 of the cases there was disease among the animals, of which 2 of them (cats) were sent to Boston.

Animals found Dead in the Neighborhood of the Cases.	
Several kittens in the street,	1
Dead cat at the corner,	1
Chickens,	4
Mice in well,	1
Rats,	1

Presence of Vermin, Insects, Rodents, etc.	Cases.
Rats and roaches,	1
Flies,	48
Bedbugs and flies,	4
Pediculi,	1
Flies and rats,	3
Roaches,	1
Rats, flies and bedbugs,	1
Bedbugs, roaches and flies,	1
Stable flies,	1

	Cases.
Ants,	1
Flies and mosquitoes,	2
Mice and flies,	1
Mice,	1
Number of children paralyzed,	61
Number of children aborted,	2
Number of adults paralyzed,	10
Number of fatal cases of children,	12
Number of fatal cases of adults,	7
Child mortality,	16.43+ per cent.
Adult mortality,	9.59+ per cent.
Total mortality for the epidemic,	26.02+ per cent.

I am greatly indebted to the physicians and the members of the local boards of health throughout my district where these cases occurred for their numerous courtesies and assistance in obtaining this information.

X.

PRINCIPLES OF THE TREATMENT OF INFANTILE PARALYSIS.[1]

ROBERT W. LOVETT, M.D., BOSTON.

The question of the treatment of infantile paralysis is in these days always before the medical profession of the United States, because each summer since 1907 has left behind it hundreds and sometimes thousands of victims, and with our added experience and our facilities for clinical observation, unfortunately far greater than anywhere else in the world, our ideas of treatment have progressed and have become more defined. If the point of view advanced in this paper is that of an orthopedic surgeon, it is because in most bad cases the surgeon or the orthopedic surgeon is sooner or later consulted, and these two from seeing end-results are perhaps best equipped to judge of the efficiency of the various forms of the earlier treatment.

EARLY DIAGNOSIS IN RELATION TO TREATMENT.

The diagnosis of the disease before the appearance of paralysis is constantly overlooked. This is a cause of dissatisfaction to the family and of mortification to the physician, but probably not of great moment to the patient, for even when a correct or a provisional diagnosis is made early it is doubtful if anything can be done to influence greatly the course of the affection. Flexner's [2] experiments with hexamethylenamin showed that it seemed to have some immunizing effect on monkeys, but that after infection had occurred it had no effect. Still, on the supposition that monkeys and children may not be affected in just the same way, and as the drug in moderation seems to be harmless, the early use of hexamethylenamin seems our best chance. Occasionally cases occur which suggest that it may have been useful. For example, in July, 1911, three children in one family were affected at intervals of three or four days with fever, prostration and gastrointestinal disturbance. The diagnosis of the first case was made only after the second child was in the

[1] Reprinted from the Journal of the American Medical Association Jan. 24, 1914, Vol. LXII., pp. 251–254.

[2] Flexner, Simon, and Clark, Paul F.: Experimental Poliomyelitis in Monkeys, The Journal A. M. A., Feb. 25, 1911, p. 585.

height of her attack and before the third was affected. The first child received no hexamethylenamin, the second had a little, and the third had large doses from the beginning. The first child was severely paralyzed from the waist down, the second had weakness of the legs and back for a few months, and the third had no muscular involvement. All were equally sick. Such an observation, of course, proves nothing, but in connection with the early history of other cases that I have analyzed has made me feel that the early use of hexamethylenamin is desirable in suspected cases.

TREATMENT OF THE ACUTE PHASE.

Little time need be spent in discussing the general measures of rest, catharsis, light feeding and quiet, which are universally agreed on. If any criticism is to be made it is that children slightly affected are often allowed to be too active; but generally the patient is so ill at first and so tender after the attack that this matter takes care of itself.

In formulating the treatment of the acute attack and of the days following we have only to remember the pathology of the affection. It is essentially a hemorrhagic myelitis with a widely distributed accompanying meningitis. Such a condition obviously demands general quiet, freedom from excitement and activity, and recumbency for a period of days or weeks. Even in the slighter cases the lesion cannot be immediately recovered from, and the observations on monkeys have taught us that some of the muscular paralysis is due to an edema in the cord accompanying the hemorrhagic process. The need of the measures spoken of must be self-evident, and my own practice has been to secure for even the slightest cases quiet for at least two or three weeks, and for the severer cases quiet until all tenderness has disappeared.

It must be said, however, that comparison of different treatments must be made with care, for no two cases are alike, the dose of poison or the individual resistance varies enormously, and the condition in the acute attack gives no very accurate means of telling what the ultimate outcome will be, except that the general conclusion may be formulated that on the whole very severe attacks are most often accompanied by severe paralysis and slighter attacks by more moderate and slighter forms.[1]

Two hundred and thirty-four cases of paralysis occurring in Massachusetts in 1907 were investigated in 1911 by a competent orthopedic surgeon.[2] Seeing as many of the patients as could be traced, he found that 25 per cent. had wholly recovered without regard to whether they had or had not been treated, and, moreover, that the analysis of the early

[1] Lovett, Robert W., and Lucas, W. P.: Infantile Paralysis, a Study of 635 Cases, The Journal A. M. A., Nov. 14, 1908, p. 1677.
[2] Wood, B. E.: Boston Med. and Surg. Jour., Oct. 5, 1911.

history of these cases showed them to be average cases and that they could not in the early stage have been picked as cases in which the patients were especially likely to recover.

All of this goes to confirm the statement that the outcome of the case is not wholly determined by the treatment received, a point which parents are slow to grasp.

Treatment of the Tender Convalescent Phase.

Any rigid division of the disease into stages is misleading and out of accord with facts, because one stage melts into another so gradually that a dividing line is impossible. Still there are different aspects of the affection, which have been spoken of here as phases.

Following the subsidence of fever the patient is generally paralyzed in one or more limbs, tender to the touch and on motion, somewhat prostrated, and generally shows the results of a general infection. The latter, however, is soon recovered from, although in the severer cases the patient is generally below par for some time. This is the period when spontaneous improvement will begin, and the family may be assured that improvement which they can see will occur in a few weeks. It is generally difficult to make the family and the practitioner who has not had much experience in the disease understand that the best treatment at this stage is to let the patient alone except for preventing contraction of the Achilles tendon, which may occur to a troublesome extent in the first two or three weeks.

So long as tenderness lasts it may be accepted as evidence of the existence of some degree of active myelitis around the motor and especially the sensory centers of the cord. Under these conditions it seems unphysiologic to stimulate by passive movements, massage or electricity the peripheral parts connected with those centers. Yet the early use of these measures is a common practice.

The tenderness may last from two to three months after the attack, and a perfectly inactive treatment is hard to pursue when the family has heard of the wonders of electricity and massage, and is anxious not to lose time. But so long as the tenderness lasts, the best practice is to let the patient alone so far as active treatment goes. Frequent changes of position are desirable, and there is no objection to the sitting position for the convalescent, to outdoor air, or to immersion in a warm bath with whatever active movement under water may be accomplished without discomfort.

There is no danger that the joints will stiffen, and in the first weeks the only troublesome complication to be feared, as has been said, is contraction of the Achilles tendon. If this is occurring it is justifiable to

stretch the posterior muscles gently with the hand, even at the risk of causing some discomfort, rather than to allow a permanent contraction to occur. The easiest means of preventing the contraction is from the beginning to have the soles of the feet rest against a box covered with a blanket placed against the bottom of the bed, which holds the feet at a right angle to the legs. Unless this is done the weight of the bed-clothes and the dropped position of the unsupported foot in sitting will in most cases cause some degree of talipes equinus. The frequency with which this complication is seen in practice shows that it is not an imaginary danger.

Treatment of the Convalescent Phase after Tenderness has gone.

With the disappearance of tenderness the time for active treatment has begun, and the sooner the patient is put on his feet and resumes activity the better. It seems probable on general principles that in cases of any degree of severity, even if tenderness disappears earlier than four weeks, active treatment should not be begun before that time, while some authorities would forbid active measures before the expiration of six weeks from the onset. In my experience, however, I have seen nothing but good come from instituting treatment at four weeks when the disappearance of tenderness has warranted it. The general condition of the patient, moreover, must not be neglected, as many of the children at this time have not wholly recovered from the effects of the infection, and are anæmic, poorly, and easily fatigued.

The therapeutic measures at our disposal are massage, electricity and muscle training.

Massage may be expected to improve the local and general circulation, to facilitate the flow of lymph, and to retard muscular deterioration. It cannot, however, be expected to facilitate the transmission of a motor impulse from the brain to the affected or weakened muscle. In estimating its value in the treatment we must remember that it is only a part of the treatment.

Electricity is less highly regarded in the treatment than was formerly the case. The unintelligent use of electricity month after month to the exclusion of other measures has been one of the handicaps which has stood in the way of the best progress in many cases. It is quite possible that it may improve the muscular condition, and it may induce the contraction of muscles which cannot be reached otherwise, but its value has apparently been overrated, and in my own practice I have seen no con-
nce that its use was beneficial, and I have preferred to
r the measures which I could see were of distinct use.

Muscle training is apparently the most useful of the three therapeutic measures mentioned, apparently because it rests on the best physiologic basis. This is what happened before the attack, and what we want to have happen again, when the child desires to contract a certain muscle to perform a certain motion: an impulse to move the muscle started from the motor area in the brain, and descended along the spinal cord motor-tracts to the spinal center controlling that muscle, where it was modified and distributed to the proper motor nerves and sent along them to cause muscular contraction. By the disease certain spinal motor centers were destroyed, and can therefore no longer act to distribute motor impulses to their muscles. But such spinal centers and their connections are complicated affairs, and every muscle is connected with several centers, every center sends impulses to more than one muscle, and, moreover, the connections between the spinal centers are many. Unless, therefore, the destruction in the cord has been a very extensive one it is likely that some of the motor centers in any one region will have escaped destruction, and that it may be possible to establish new connections around the destroyed centers. If a railway wreck occurs on the main line and the track is blocked it is often possible to send trains by means of a branch line around the obstruction, so that service between the terminals is maintained. In the same way after a wreck of certain nerve centers it may be possible by a modified route to send a motor impulse from brain to muscle. On this principle of establishing new connections and opening new paths rests most of the claim of muscle training, but not entirely on this, because on account of the local edema of the cord accompanying the infiltration and hemorrhagic process certain motor centers are temporarily put out of order but not destroyed. Muscle training aids these to recover function.

Muscle training in its most obvious form consists in aiding the patient to perform a certain movement with the hope of stimulating an impulse from the brain to the weakened or paralyzed muscles. If, for instance, the dorsal flexors of the foot do not act, through being stretched, weakened, partly paralyzed or wholly paralyzed, in the exercise the foot is dorsally flexed with the hand and the patient directed to assist. If there is any muscular response less and less aid is given to the muscle by the hand, and it may be that in this way it can be trained to perform its function. A detailed account of the exercises for the different groups of muscles has been given by Wright.[1]

In my personal experience the success of muscle training and other measures has been greater in the legs than in the arms.

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[1] Wright: Muscle Training in the Treatment of Infantile Paralysis, Boston Med. and Surg. Jour., Oct. 24, 1912.

Another and equally useful form of muscle training consists in getting the patient on his feet at the earliest possible moment in order to call forth the instinctive muscular actions induced by the efforts to walk and balance. Even before it is possible to make much progress in this way, sitting is useful for the spinal and trunk muscles.

The Use of Apparatus and Braces.

Many patients at the beginning are unable to stand without apparatus, because, for example, the knees flex on account of weakness or paralysis of the quadriceps muscle. In these cases a caliper splint should be applied to hold the knees straight.[1] If the feet roll in or out varus or valgus braces should be applied. If the spine or abdomen is involved, a corset or jacket should be worn. Crutches are at first necessary in cases of paralysis of both legs. In other words, if the standing position induces malposition, such malposition must be corrected, because nothing but harm can come of it.

The fear that the early use of apparatus will promote muscular atrophy is wholly unreasonable, because nothing is so bad as disuse, and braces should mean the upright position, and the upright position means more muscular activity. The best way to avoid wearing a brace permanently is to put it on early and keep it on as long as necessary. For a growing child to walk about with a malposition is to bid for a permanent deformity.

Deformity.

If fixed deformity exists, it must be removed before treatment of any sort can be satisfactory. By fixed deformity is meant a condition in which the functions of a joint are limited, in which its arc is restricted. If a child sits with the foot dropped and it can be normally flexed by the hand to its normal extent it is merely a malposition; if it cannot be brought to a right angle it is a deformity. The difference is an important practical one.

The common and most troublesome deformities in neglected cases are fixed contractions of the feet in varus, valgus or equinus, flexion of the knees and contraction of the fascia lata, causing contraction of the thighs on the body. These deformities are easily remedied by stretching or cutting, and this is an essential preliminary to other mechanical or operative treatment. One should, however, be careful about cutting the Achilles tendon without some further operation when the anterior leg muscles are paralyzed, as an unsatisfactory limp and powerless foot may result.

Treatment in Infantile Paralysis, Department of Orthopedic Surgery, Harvard Medical Med. and Surg. Jour., June 30, 1910.

The most troublesome deformity of all is lateral curvature of the spine, which occurs to some degree in most cases in which the shoulder, back or respiratory muscles are involved, which should always be watched for. Many errors would be avoided if every child with infantile paralysis were stripped and examined for scoliosis. It is singularly resistant to treatment, and when it affects the cervicodorsal region is practically not to be controlled. Lower down in the spine it may be greatly benefited by proper jackets and exercises. But such jackets must be applied early and worn persistently, because the cause, the unilateral muscular paralysis, is always present to aggravate the condition.

Operative Treatment.

It is my purpose in this paper to give only the briefest possible outline of operative treatment. Operations are undertaken: —

1. To correct fixed deformity.
2. To improve muscular function.
3. To secure stability of useless joints.

(1) The correction of fixed deformity has already been dealt with.

(2) The improvement of muscular function is accomplished by the transference of the tendinous end of a sound muscle to the place of insertion of a paralyzed muscle, so that its function is substituted for the lost function. The operation is useful in carefully selected cases, and must rest on careful anatomic study. The technic is of great importance, and has been fully discussed.[1]

The operation should not be performed until two or three years after the acute attack, and is not suitable for very young children. The passage of a considerable length of time since the attack is not a barrier to operation, and it is useful in adults.

The most important recent additions to our knowledge are that insertion into bone or periosteum (preferably the former) is preferable to insertion into tendons, and that silk elongation of the tendon may be performed with impunity and makes the operation much more widely applicable. Good function in the operated limb cannot be expected under six months.

(3) The stability of flail and useless joints may be secured in several ways.

Arthrodesis, or the production of artificial ankylosis, in such a joint is obtained by opening the joint and removing the articular cartilage. In the ankle it has been most used, and under proper conditions furnishes a stiff ankle so that the patient need not wear a brace. It is, however, not

[1] Lange, F.: The Orthopedic Treatment of Infantile Paralysis. Am. Jour. Orthop. Surg., August, 1910: Ergebnisse der Chirurgie und Pathologie, Payr and Küttner, Berlin, 1911, II. 1.

to be done unreservedly. There is no use in stiffening an ankle unless the knee is good and can bear weight, and it is not to be done in children under 12, or a serious deformity may follow, incident to the growth of the foot, which may cause a bad varus. Arthrodesis of the knee should never be attempted in children on account of the danger of interfering with epiphyseal growth, and most adults prefer a brace which can be unlocked for bending when sitting down, to a permanently stiff leg. At the hip it is an operation little used, and chiefly to be considered when the hip is dislocated and can be reduced. In the shoulder it is sometimes useful.

Silk ligaments at the anterior aspect of the ankle are much to be preferred to arthrodesis in cases of flail ankle, because they do not cause a stiff joint, but allow dorsal flexion while they check plantar flexion or dropping of the foot; they do not cause bony distortion, and they may be safely used in middle childhood. Just as the silk elongation of a muscle is coated with fibrous tissue and becomes a tendon, so does silk inserted to check joint motion become coated with fibrous tissue to form a ligament.

In the technic one or two matters are of much importance. The crest of the tibia above the ankle is exposed, periosteum incised and turned back, and a hole drilled in the bone. One of the bones in the tarsus at the inner or outer side of the foot, or both, as the indication for support determines, is drilled in the same way, and one or two strands of heavy silk are passed through the tibial hole under the annular ligament, through the tarsal hole, and tied. The knot must come in the upper wound, as otherwise it is likely to chafe through from pressure of the boot. At least three months' support is necessary, and probably more.

The application of silk ligaments to other joints has not been fully worked out. In my own practice I have not done an arthrodesis of the ankle for dropped foot for several years, because the results of the silk ligament operation are so much more satisfactory. Other operations for fixation are advocated.[1]

In cases of paralytic calcaneus deformity, the Whitman operation[2] consists in removing the astragalus and setting the foot back in relation to the leg. After the operation the foot should be placed in the equinus position.

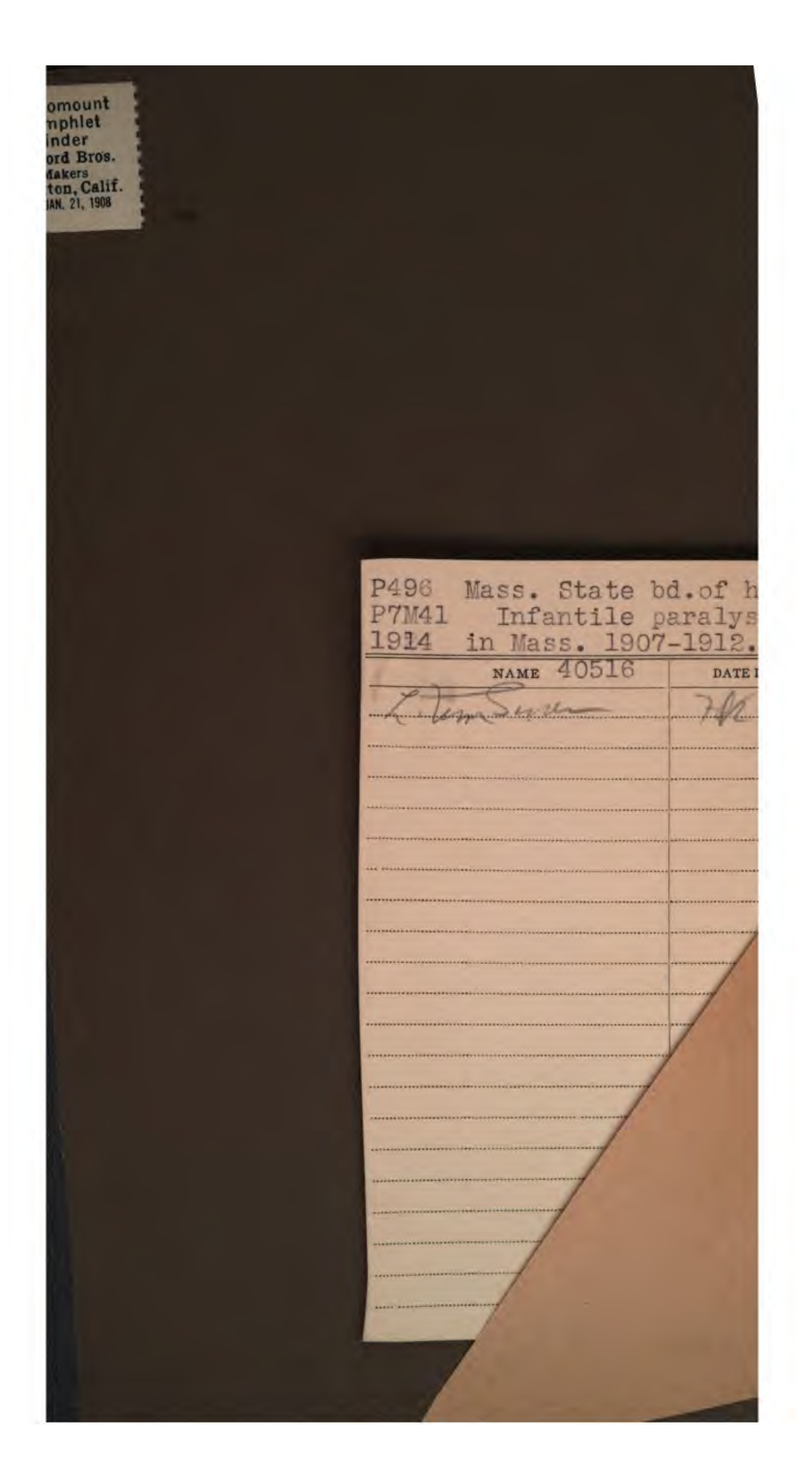
[1] Putti: Bull. d. Sc. Méd., lxxxiii, Series 8, 1912, xii. Bartow and Plummer: Buffalo Med. Jour., January, 1912.

[2] Whitman: Treatise on Orthopedic Surgery, ed. 4, Lea and Febiger, Philadelphia.

Conclusion.

The foregoing seems to me to be a summary of the modern treatment of infantile paralysis, colored of course by my own personal experience. I think that those of us who live in communities which have been severely affected become every year more hopeful and more persistent in our efforts. There seems no time limit as to improvement, and many of the most neglected cases seem the most striking when they come to proper treatment. I have merely tried to show that a treatment based on our more recent knowledge of the disease differs in some respects from our former ideas, which were based on smaller experience and a less exact pathology.





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